

# SEARCH REQUEST FORM

126419

69826

Requestor's

Name:

*Patent Code*

Serial

Number:

*10/061035*

Date:

*7/6/04*

Phone:

*Rem 4 C 70*

Art Unit:

*161V*

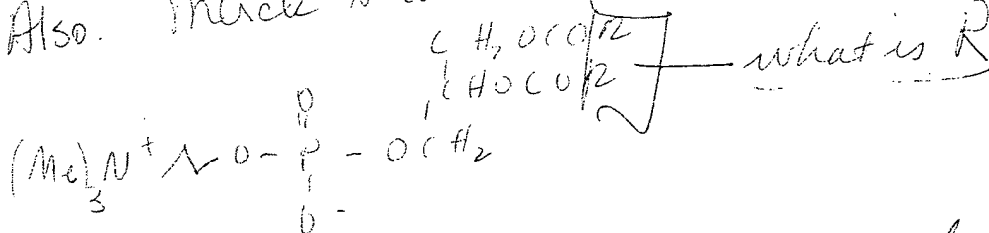
## Search Topic:

Please write a detailed statement of search topic. Describe specifically as possible the subject matter to be searched. Define any terms that may have a special meaning. Give examples or relevant citations, authors, keywords, etc., if known. For sequences, please attach a copy of the sequence. You may include a copy of the broadest and/or most relevant claim(s).

*follow up*

*Please search metabolism of  
lecithin in body. Is it  
metabolized to phosphatidyl  
choline*

*Also. Much shows lecithin as*



*Can it be acid fragments listed in claim 10?*  
*Thank u*  
*Patent*

## STAFF USE ONLY

Date completed: \_\_\_\_\_

Searcher: \_\_\_\_\_

Terminal time: \_\_\_\_\_

Elapsed time: \_\_\_\_\_

CPU time: \_\_\_\_\_

Total time: \_\_\_\_\_

Number of Searches: \_\_\_\_\_

Number of Databases: \_\_\_\_\_

### Search Site

\_\_\_\_\_ STIC

\_\_\_\_\_ CM-1

\_\_\_\_\_ Pre-S

### Type of Search

\_\_\_\_\_ N.A. Sequence

\_\_\_\_\_ A.A. Sequence

\_\_\_\_\_ Structure

\_\_\_\_\_ Bibliographic

### Vendors

\_\_\_\_\_ IG

*File 11* STN

\_\_\_\_\_ Dialog

\_\_\_\_\_ APS

\_\_\_\_\_ Geninfo

\_\_\_\_\_ SDC

\_\_\_\_\_ DARC/Questel

\_\_\_\_\_ Other

# The 2 lecithin derivatives in Registry that contain structures

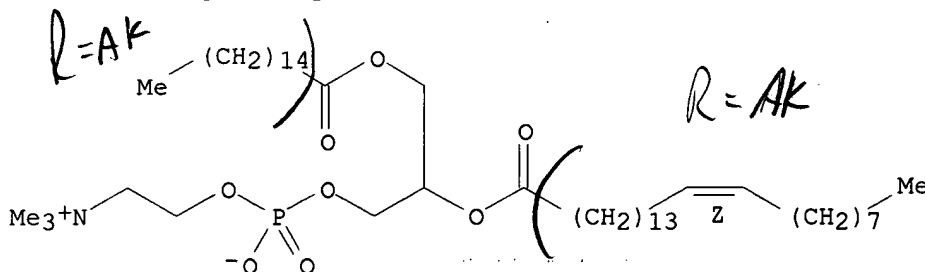
Cook 10/068,035

July 9, 2004

L3 ANSWER 1 OF 2 REGISTRY COPYRIGHT 2004 ACS on STN  
 RN 13962-36-2 REGISTRY  
 CN Choline, dihydrogen phosphate (ester), monoester with 1-palmito-2-(15-tetracosenoin), (Z)- (8CI) (CA INDEX NAME)  
 OTHER CA INDEX NAMES:  
 CN Choline, phosphate, ester with 1-palmitin 2-(cis-15-tetracosenoate) (7CI)  
 OTHER NAMES:  
 CN **Lecithins,  $\beta$ -nervonyl- $\gamma$ -palmitoyl- $\alpha$ -**  
 FS STEREOSEARCH  
 MF C48 H94 N O8 P  
 LC STN Files: CAOLD

*R = fatty acids  
in Merck*

Double bond geometry as shown.



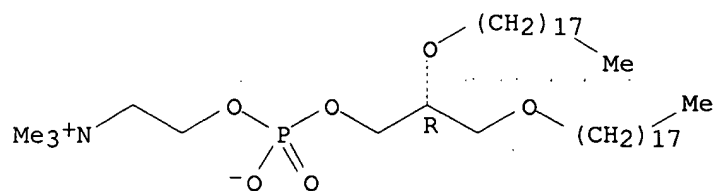
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L3 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2004 ACS on STN  
 RN 1188-85-8 REGISTRY  
 CN 3,5,9-Trioxa-4-phosphaheptacosan-1-aminium, 4-hydroxy-N,N,N-trimethyl-7-(octadecyloxy)-, inner salt, 4-oxide, (7R)- (9CI) (CA INDEX NAME)  
 OTHER CA INDEX NAMES:  
 CN 3,5,9-Trioxa-4-phosphaheptacosan-1-aminium, 4-hydroxy-N,N,N-trimethyl-7-(octadecyloxy)-, inner salt, 4-oxide, (R)-  
 OTHER NAMES:  
 CN Glycerol-3-sn-phosphorylcholine 1,2-bis(octadecyl ether)  
 CN **Lecithin distearyl ether**  
 FS STEREOSEARCH  
 MF C44 H92 N O6 P  
 LC STN Files: BEILSTEIN\*, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, TOXCENTER, USPATFULL

(\*File contains numerically searchable property data)

DT.CA Caplus document type: Journal; Patent  
 RL.P Roles from patents: BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)  
 RL.NP Roles from non-patents: BIOL (Biological study); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)  
 RLD.NP Roles for non-specific derivatives from non-patents: BIOL (Biological study); PROC (Process)

Absolute stereochemistry.



\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\*

20 REFERENCES IN FILE CA (1907 TO DATE)  
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA  
20 REFERENCES IN FILE CAPLUS (1907 TO DATE)  
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

[Go Back](#) | [Nutrition for a Living Planet - Home Page](#)

## Phosphatidylcholine (PC)

Phosphatidylcholine (PC) is a purified extract from lecithin and is one of the components of bile (it is required for normal transport of bile acids). Emulsifies and breaks down fat deposits in the body, which make it helpful in the prevention of gallstones, atherosclerosis, heart disease, and liver problems. Research has shown that Phosphatidylcholine is beneficial in depression, memory loss and neurological disorders. It is 3 times more potent than Lecithin

Phosphatidylcholine is one of the most important support nutrients for the liver. PC is a phospholipid, a large biological molecule that is a universal building block for cell membranes. A cell's membranes are its essence: they regulate the vast majority of the activities that make up life. Most liver metabolism occurs on cell membranes, which occupy about 33,000 square meters in the human. More than 2 decades of clinical trials indicate that PC protects the liver against damage from alcoholism, pharmaceuticals, pollutant substances, viruses, and other toxic influences, most of which operate by damaging cell membranes. The human liver is confronted with tens of thousands of exogenous substances. The metabolism of these xenobiotics can result in the liver's detoxicative enzymes producing reactive metabolites that attack the liver tissue. Dietary supplementation with PC (a minimum 800 mg daily, with meals) significantly speeds recovery of the liver. PC has also been shown to be effective against alcohol's liver toxicity in well-controlled studies on baboons. PC has other qualities that enhance its usefulness as a dietary supplement. PC is safe, and is a safer means for dietary choline repletion than choline itself. PC is fully compatible with pharmaceuticals, and with other nutrients. PC is also highly bioavailable (about 90% of the administered amount is absorbed over 24 hours), and PC is an excellent emulsifier that enhances the bioavailability of nutrients with which it is co-administered. PC's diverse benefits and proven safety indicate that it is a premier liver nutrient.

Lecithin is a nutrient compound which was first isolated from egg yoke in 1850 by Maurice Bobley. Since that time, it has been shown to be present in many foods. Soybeans and other legumes, grains, wheat germ, brewers yeast, and fish, as well as egg yokes are all good sources of lecithin.

Biochemically speaking, lecithin belongs to a group of nutrients known as lipids (fats, oils, waxes) and is a phospholipid called phosphatidylcholine. It is important to note that since what is commercially called lecithin is actually only one-third true lecithin. The other two-thirds is made up of other phospholipids.

Lowering serum cholesterol levels has been recommended as an important factor in coronary health. Lecithin, specifically granular lecithin with 98%+ phosphatidylcholine content, can be a valuable component in that process.

In 1958, that Dr. Lester M. Morrison, director of a research unit at Los Angeles County General Hospital, first published (Geriatrics, January, 1958) his findings that lecithin could be used to lower cholesterol levels. In fact, Dr. Morrison reported that "lecithin was found to be the most effective cholesterol lowering agent tested." He reported that 80% of his patients suffering from high serum cholesterol levels showed an average decrease of 41% in serum cholesterol after taking lecithin for several weeks.

Instead of "blocking" absorption of cholesterol in the digestive tract as other cholesterol reducing agents did, lecithin enhanced the metabolism of cholesterol in the digestive system and aided in its transport

through the circulatory system. The lecithin acted as an emulsifier and broke down the fats and cholesterol in the diet into tiny particles and held them in suspension, preventing them from sticking to blood platelets or arterial walls. It is when fats are not properly emulsified, that they become "sticky" and this is the major cause of blood clots, atherosclerosis, and coronary thrombosis. Interestingly enough, researchers have since demonstrated that atherosclerosis (blockage of the arteries) can be induced in the laboratory by either increasing the cholesterol introduced into the body or by decreasing lecithin intake.

Researchers Adams and Morgan have also shown that lecithin from a vegetable source (soybeans) is more effective than lecithin from an animal source (eggs) in accelerating re-absorption of cholesterol back into the blood stream that has adhered to the walls of blood vessels and caused blockage.

This difference is attributed to the fact that lecithin from animal sources contains high amounts of saturated fatty acids, while lecithin from vegetable sources are about 80% unsaturated fatty acids.

Perhaps the most interesting new findings on lecithin concern its connection with the functioning of the brain and nervous system.

The main source of energy for the brain comes from a combination of oxygen and glucose (sugar). For brain cells to function normally they must receive a constant supply of these nutrients. As areas of the brain become more active blood flow into and out of these areas increase.

In addition to oxygen and glucose, the brain uses chemical compounds known as phospholipids. These phospholipids make up the covering of nerve cells that assist in the transfer of information from cell to cell. Without phospholipids brain cell activity may become abnormal and cause problems in the nervous system.

Certain diseases like Alzheimer's disease and brain tumors can affect blood flow to the brain and change the way the brain metabolizes phospholipids. In addition to diseases, changes in the brain occur with normal healthy aging.

Besides being an important factor in controlling cholesterol levels and aiding coronary health, lecithin is involved in a myriad of body functions. Every cell of your body contains lecithin. Lecithin is responsible for maintaining the surface tension of the cell membrane. It therefore controls what goes in and out of each cell, allowing nutrients in, or wastes out. Without enough lecithin, the cell wall hardens, thus not allowing enough nutrients in or wastes out. This means premature aging of cells. The surface tension of the cell maintained by lecithin is also responsible for transmitting nerve impulses and messages through or from the cell.

A key factor in proper brain and nerve transmissions is the presence of cellular substance called acetylcholine. Acetylcholine deficiencies are linked with the neurological disorders tardive dyskinesia (involuntary facial grimaces and body jerking), Huntington's chorea, Friedrich's ataxia (speech impairment, irregular movements, and paralysis), olivaponto-cerebellar atrophy (wasting away of the brain), Alzheimer's disease (a mind destroying disease that starts with memory difficulties), and myasthenia gravis (progressive paralysis).

Until recently, medical researchers were using choline chloride to help their patients who suffered from these insidious brain disorders to produce more acetylcholine in their bodies. However, in 1977, Dr. Richard Wurtman and his colleagues at Massachusetts Institute of Technology found that lecithin (which contains phosphatidylcholine) increased serum choline levels more than three times as much as

the previously used choline chloride and kept those levels raised more than three times as long. This meant that researchers had found a way to significantly raise acetylcholine levels in their patients since acetylcholine production in the brain was dependent on serum choline levels.

An unexpected discovery by researchers at The National Institutes of Health (NIH), may help to explain how Alzheimer's disease causes memory loss. The research shows that beta amyloid, a common protein in the brain, can make cell membranes leak choline, and thus reduce production of acetylcholine in cells. Choline, an essential ingredient in acetylcholine, has been known for many years to help store and retrieve memories. Two hallmarks of Alzheimer's disease are accumulation in the brain of beta amyloid and reduction of the concentration of acetylcholine. In Alzheimer's disease, as well as in older subjects with Down syndrome, the brain cells which produce acetylcholine are known to die.

The research is reported in the May 23rd issue of Brain Research by investigators at the National Institute on Aging (NIA) and the National Institute of Neurological Disorders (NINDS). According to Dr. Stanley Rapoport, Chief of the NIA's Laboratory of Neurosciences, "We think that increased leakage of choline through the nerve cell membranes, due to prolonged exposure to excess concentrations of beta amyloid, may make these cells more vulnerable. This could contribute to the symptoms of Alzheimer's disease and Down's syndrome dementia."

Studies on the effect of phosphatidylcholine administration on memory are limited. We administered egg phosphatidylcholine to mice with dementia and to normal mice and compared the differences in memory and serum choline concentration, and choline and acetylcholine concentrations and choline acetyltransferase activities of three forebrain regions (cortex, hippocampus and the remaining forebrain). Mice with dementia were produced by mating sibling mice who had impaired memory for > 20 generations. These mice had poor memory and low brain acetylcholine concentration. We administered 100 mg of egg phosphatidylcholine (phosphatidylcholine group) or water (control group) by gavage to each mouse daily for about 45 d. Control mice with dementia had poorer memory in passive avoidance performance and lower brain choline (cortex and hippocampus) and acetylcholine (hippocampus and forebrain excluding cortex and hippocampus) concentrations and lower cortex choline acetyltransferase activity than the control normal mice ( $P < 0.05$ ). The administration of phosphatidylcholine to mice with dementia improved memory and generally increased brain choline and acetylcholine concentrations to or above the levels of the control normal mice. In normal mice, phosphatidylcholine treatment did not affect memory or acetylcholine concentrations in spite of the great increase in choline concentrations in the three brain regions. Serum choline concentration in mice treated with phosphatidylcholine increased to a similar level in both strains of mice, indicating that the absorption of phosphatidylcholine was not impaired in mice with dementia. The results suggest that administration of egg phosphatidylcholine to mice with dementia increases brain acetylcholine concentration and improves memory.

- 
- Vikki McInnis-Shaw. Liver Disease Medical Glossary. Hepatitis Central.
  - Parris M. Kidd, Ph.D. Phosphatidylcholine: A Superior Protectant Against Liver Damage. *Alt Med Rev* 1996;1(4):258-274
  - Hanin I, Ansell GB: *Lecithin: Technological, Biological, and Therapeutic Aspects*. New York: Plenum Press, 1987
  - Positron Emission Tomography Imaging of Human Brain Phospholipid Metabolism in Relation to Age and Disease. National Institute of Neurological Disorders and Stroke 94-N-0205
  - Hypothesis for Cause of Memory Loss in Alzheimer's Disease Proposed. National Institute on Aging.
  - Chung SY, Moriyama T. Et al. Administration of phosphatidylcholine increases brain acetylcholine concentration and improves memory in mice with dementia. *J Nutr*, 125(6) :1484-9

1995 Jun

=&gt; d que 133

L24 5404 SEA FILE=HCAPLUS ABB=ON PLU=ON "FATTY ACIDS (L) POLYUNSATD.,  
N-3"+OLD/CT  
L30 36 SEA FILE=HCAPLUS ABB=ON PLU=ON PHOSPHATIDYLCHOLINES+NT/CT(L) "  
Ω-3"  
L31 30 SEA FILE=HCAPLUS ABB=ON PLU=ON L30 AND L24  
L32 3 SEA FILE=HCAPLUS ABB=ON PLU=ON Ω-3 PHOSPHATIDYL?  
L33 31 SEA FILE=HCAPLUS ABB=ON PLU=ON L31 OR L32

=&gt; d l33 ibib abs hitind 1-31

L33 ANSWER 1 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2004:219834 HCAPLUS

DOCUMENT NUMBER: 140:247039

TITLE: Lipids conjugated to ω-3 fatty acids and  
anticancer drugs, preparation, and methods of use  
INVENTOR(S): Stillwell, William; Zerouga, Mustapha; Jensi, Laura  
J.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 20 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	US 2004052837	A1	20040318	US 2003-602978	20030624
PRIORITY APPLN. INFO.:				US 2002-392117P P	20020627
AB	Lipid based antiproliferative agents and methods of use thereof are disclosed. Antiproliferative compns. of the invention comprise a lipid conjugated to an ω-3 fatty acid and a chemotherapeutic agent, e.g. phosphatidylcholine conjugated to docosahexaenoic acid and methotrexate. Preparation of the conjugates is described.				
IC	ICM A61K009-127				
	ICS A61K031-525				
NCL	424450000; 514560000; 514251000				
CC	1-6 (Pharmacology)				
	Section cross-reference(s): 29, 63				
IT	Lipids, biological studies				
	<b>Phosphatidylcholines, biological studies</b>				
	Phosphatidylethanolamines, biological studies				
	RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)				
	(conjugates; lipids conjugated to ω-3 fatty acids and anticancer drugs, preparation, and methods of use)				
IT	<b>Fatty acids, biological studies</b>				
	RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)				
	(polyunsatd., n-3, conjugates; lipids conjugated to ω-3 fatty acids and anticancer drugs, preparation, and methods of use)				

L33 ANSWER 2 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:99433 HCAPLUS

DOCUMENT NUMBER: 138:250469



TITLE: Effect of phospholipids containing  $\omega$ -3 fatty acids on the structural changes in microsomal membranes from functionally distinct cells

AUTHOR(S): Datsenko, Z. M.; Krivenko, O. M.; Nechitailo, L. O.; Shovkun, S. A.; Khmel, T. O.; Perederii, O. F.

CORPORATE SOURCE: Inst. Biokhim. im. O. V. Palladina, NAN Ukr., Kiev, Ukraine

SOURCE: Ukrain'skii Biokhimichnii Zhurnal (2002), 74(4), 44-49  
CODEN: UBZKAA

PUBLISHER: Institut Biokhimii im. O. V. Palladina NAN Ukraini

DOCUMENT TYPE: Journal

LANGUAGE: Ukrainian

AB As a result of the exptl. researches conducted it has been shown that administration of some normal animal marine phospholipids (PL) including in their structure  $\omega$ -3 polyunsatd. fatty acids (PUFA) provides for quant. changes of individual PL, fatty acids (FA) content and quantity in general and individual PL of liver, heart, brain and gonads microsomes. While estimating general microsomal PL fraction FA content under the action of PL  $\omega$ -3 PUFA FA concentration change, unsatn. index ( $\omega$ 6/ $\omega$ 3) and relation of arachidonic acid to docosahexaenoic (AA/DHA) decrease have been identified. The decrease of AA/DHA relationship occurs due to AA and DHA quant. changes. In the case of AA increase in some tissues there is observed the decrease of docosapentaenoic acid and increase of DHA and eicosapentaenoic (EPA) acids. As a result of studying FA content in the individual PL composition it has been identified that certain PL classes characteristic for some tissues respond by changes of some certain FA. The relationship  $\omega$ 6/ $\omega$ 3 has been shown as decreasing in phosphatidylcholine (PC) all tissues microsomes (liver, gonads, heart, brain), in phosphatidylethanolamine (PEA) of liver and cardiac microsomes, in phosphatidylserine (PS) this relationship decreases in the liver, brain and heart, for phosphatidylinositol (PI) the changes take place in liver, gonads, brain. Simultaneously, the decrease of AA/DHA relationship in the individual PL decrease of AA and increase of EPA and DHA depend on the tested tissues. The marine phospholipids might be supposed to render their effect on AA metabolism resulting in AA/DHA relationship in PEA and PS relationship displays itself as specific and depends on the tissues functions. The preference of PEA and PS use by certain tissues microsomes could be explained by their membrane protective capability.

CC 6-5 (General Biochemistry)

Section cross-reference(s): 13

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines, biological studies

Phosphatidylinositols

Phosphatidylserines

Phospholipids, biological studies

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(effect of phospholipids containing  $\omega$ -3 fatty

acids on structural changes in microsomal membranes from functionally distinct cells)

IT **Fatty acids, biological studies**

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(polyunsatd., n-3; effect of

phospholipids containing  $\omega$ -3 fatty acids on structural changes in microsomal membranes from functionally distinct cells)

L33 ANSWER 3 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:42832 HCAPLUS

DOCUMENT NUMBER: 138:112446

TITLE: Omega-3 fatty acids in the treatment of depression

INVENTOR(S): Stoll, Andrew  
 PATENT ASSIGNEE(S): USA  
 SOURCE: U.S. Pat. Appl. Publ., 7 pp., Cont.-in-part of U.S.  
 6,344,482.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003012827	A1	20030116	US 2002-83913	20020227
WO 9739759	A2	19971030	WO 1997-US6712	19970423
WO 9739759	A3	19980115		
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6344482	B1	20020205	US 1999-269361	19990322
WO 2003072111	A2	20030904	WO 2003-US5926	20030227
WO 2003072111	A3	20040318		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:  
 WO 1997-US6712 W 19970423  
 US 1999-269361 A2 19990322  
 US 1996-16140P P 19960424  
 US 2002-83913 A 20020227

AB The present invention is directed to a method of treating patients with major depression by administering  $\omega$ -3 fatty acids. These may be administered in a substantially purified form, as part of a pharmaceutical composition, or as part of a larger mol., e.g., a triacylglycerol, which releases free fatty acid after ingestion by a patient. The present invention is also directed to triacylglycerols which are esterified at the  $\gamma$ -carbon of glycerol to phosphatidylcholines and at either the  $\alpha$ - or  $\beta$ -carbon of glycerol to an  $\omega$ -3 fatty acid. These "**omega.-3 phosphatidylcholines**" are also used in the treatment of patients with major depression.

IC ICM A61K035-78  
 ICS A61K033-00; A61K031-685; C07F009-02; A61K031-5513  
 NCL 424722000; 514221000; 424730000; 514078000; 514560000; 554080000  
 CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 1

IT **Fatty acids, biological studies**

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (polyunsatd., n-3; omega-3 fatty acids in treatment of depression)

IT **Phosphatidylcholines, biological studies**

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (unsatd.; omega-3 fatty acids in treatment of depression)

L33 ANSWER 4 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 2002:451307 HCAPLUS  
DOCUMENT NUMBER: 137:153186  
TITLE: In vitro mimicry of essential fatty acid deficiency in human endothelial cells by TNF $\alpha$  impact of  $\omega$ -3 versus  $\omega$ -6 fatty acids  
AUTHOR(S): Mayer, Konstantin; Schmidt, Reinhold; Muhly-Reinholz, Marion; Bogeholz, Tina; Gokorsch, Stephanie; Grimminger, Friedrich; Seeger, Werner  
CORPORATE SOURCE: Medizinische Klinik II, Zentrum fur Innere Medizin, Justus-Liebig-University, Giessen, D-35392, Germany  
SOURCE: Journal of Lipid Research (2002), 43(6), 944-951  
CODEN: JLPRAW; ISSN: 0022-2275  
PUBLISHER: Lipid Research, Inc.  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Severe endothelial abnormalities are a prominent feature in sepsis with cytokines such as tumor necrosis factor (TNF) $\alpha$  being implicated in the pathogenesis. As mimic to inflammation, human umbilical vascular endothelial cells (HUVEC) were incubated with TNF $\alpha$  for 22 h, in the absence or presence of the  $\omega$ -6 fatty acid (FA), arachidonic acid (AA), or the alternative  $\omega$ -3 FA eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). TNF $\alpha$  caused marked alterations in the PUFA profile and long chain PUFA content of total phospholipids (PL) decreased. In contrast, there was a compensatory increase in mead acid [MA, 20:3( $\omega$ -9)], the hallmark acid of the essential fatty acid deficiency (EFAD) syndrome. Corresponding changes were noted in phosphatidylcholine, phosphatidylethanolamine, phosphatidylserine, and phosphatidylinositol, but not in the sphingomyelin fraction. Supplementation with AA, EPA, or DHA markedly increased the resp. FA contents in the PL pools, suppressed the increase in MA, and resulted in a shift either toward further predominance of  $\omega$ -6 or predominance of  $\omega$ -3 FA. We conclude that short-term TNF $\alpha$  incubation of HUVEC causes an EFAD state hitherto only described for long-term malnutrition, and that endothelial cells are susceptible to differential influence by  $\omega$ -3 vs.  $\omega$ -6 FA supplementation under these conditions.  
CC 14-3 (Mammalian Pathological Biochemistry)  
Section cross-reference(s): 15, 18  
IT **Phosphatidylcholines, biological studies**  
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)  
(in vitro mimicry of essential fatty acid deficiency in human endothelial cells by TNF $\alpha$  impact of  $\omega$ -3 vs.  $\omega$ -6 fatty acids in relation to inflammation and sepsis)  
IT **Fatty acids, biological studies**  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(polyunsatd., n-3; in vitro mimicry of essential fatty acid deficiency in human endothelial cells by TNF $\alpha$  impact of  $\omega$ -3 vs.  $\omega$ -6 fatty acids in relation to inflammation and sepsis)  
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 5 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 2000:323466 HCAPLUS  
DOCUMENT NUMBER: 133:42712  
TITLE: Effect of  $\alpha$ -tocopherol and phospholipids with  $\omega$ -3 polyunsaturated fatty acids to the membrane properties

AUTHOR(S): Krivenko, O. M.  
 CORPORATE SOURCE: Inst. Biokhim. im. O. V. Palladin, NAN Ukr., Kiev, Ukraine  
 SOURCE: Ukrainskii Biokhimicheskii Zhurnal (1999), 71(5), 127-131  
 CODEN: UBZHD4; ISSN: 0201-8470  
 PUBLISHER: Institut Biokhimii im. A. V. Palladina NAN Ukrainy  
 DOCUMENT TYPE: Journal  
 LANGUAGE: Ukrainian

AB As a result of the investigations conducted it was shown that  $\alpha$ -tocopherol and phospholipids with  $\omega$ -3 polyunsatd. fatty acids influence differently the lipid composition of heart microsomal membranes. Vitamin E deficiency caused the increase of lysophospholipids (lysophosphatidylcholine and lysophosphatidylethanolamine) and that of diphosphatidylglycerol as well as the tendency to sphingomyelin and phosphatidylethanolamine decrease in cardiac microsomes. Administration with food of both  $\alpha$ -tocopherol and the complex of phospholipids with  $\omega$ -3 polyunsatd. fatty acids from marine animals stabilized the content of phospholipids in microsomal membranes. Marine phospholipid complex was more effective than  $\alpha$ -tocopherol. Administration of phospholipids with  $\omega$ -3-fatty acids during the period of 30 days increased the polyunsatd. fatty acid:saturated fatty acid ratio in cardiac microsomal membranes. Besides, administration of phospholipids increased the levels of eicosapentaenoic and docosahexaenoic acids in microsomal membranes. Thus, vitamin E deficiency changes the ratio between membrane phospholipids and fatty acids, resulting in disturbances of membrane permeability and function. The neg. effect of E-deficiency on the membrane can be corrected by the administration of marine phospholipids with  $\omega$ -3 polyunsatd. fatty acids.

CC 18-2 (Animal Nutrition)  
 Section cross-reference(s): 6, 13

IT Cardiolipins

**Lysophosphatidylcholines**

Lysophosphatidylethanolamines

Sphingomyelins

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(effect of  $\alpha$ -tocopherol and phospholipids with  $\omega$ -3 polyunsatd. fatty acids on lipid composition of heart microsomal membranes)

IT **Fatty acids, biological studies**

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(polyunsatd., omega-3; effect of  $\alpha$ -tocopherol and phospholipids with  $\omega$ -3 polyunsatd. fatty acids on lipid composition of heart microsomal membranes)

L33 ANSWER 6 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2000:128258 HCAPLUS

DOCUMENT NUMBER: 132:232177

TITLE: Changes in phosphatidylcholine fatty acid composition are associated with altered skeletal muscle insulin responsiveness in normal man

AUTHOR(S): Clore, John N.; Harris, Paul A.; Li, Jing; Azzam, Amin; Gill, Ranjodh; Zuelzer, Wilhelm; Rizzo, William B.; Blackard, William G.

CORPORATE SOURCE: Departments of Internal Medicine, Surgery, and

Pediatrics, Virginia Commonwealth University,  
Richmond, VA, 23298, USA

SOURCE: Metabolism, Clinical and Experimental (2000), 49(2),  
232-238  
CODEN: METAAJ; ISSN: 0026-0495

PUBLISHER: W. B. Saunders Co.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The fatty acid composition of skeletal muscle cell membrane phospholipids (PLs) is known to influence insulin responsiveness in man. We have recently shown that the fatty acid composition of phosphatidylcholine (PC), and not phosphatidylethanolamine (PE), from skeletal muscle membranes is of particular importance in this relationship. Efforts to alter the PL fatty acid composition in animal models have demonstrated induction of insulin resistance. However, it has been more difficult to determine if changes in insulin sensitivity are associated with changes in the skeletal muscle membrane fatty acid composition of PL in man. Using nicotinic acid (NA), an agent known to induce insulin resistance in man, 9 normal subjects were studied before and after treatment for 1 mo. Skeletal muscle membrane fatty acid composition of PC and PE from biopsies of vastus lateralis was correlated with insulin responsiveness using a 3-step hyperinsulinemic-euglycemic clamp. Treatment with NA was associated with a 25% increase in the half-maximal insulin concentration ([ED50] 52.0 to 64.6  $\mu$ U/mL), consistent with decreased peripheral insulin sensitivity. Significant changes in the fatty acid composition of PC, but not PE, were also observed after NA administration. An increase in the percentage of 16:0 (21% to 21.7%) and decreases in 18:0 (6.2% to 5.1%), long-chain n-3 fatty acids (1.7% to 1.4%), and total polyunsatd. fatty acids ([PUFAs] 8.7% to 8.0%) are consistent with a decrease in fatty acid length and unsatn. in PC following NA administration. The change in ED50 was significantly correlated with the change in PUFAs ( $r = -.65$ ). These studies suggest that the induction of insulin resistance with NA is associated with changes in the fatty acid composition of PC in man.

CC 2-6 (Mammalian Hormones)

IT Fatty acids, biological studies  
RL: BOC (Biological occurrence); BSU (Biological study, unclassified);  
BIOL (Biological study); OCCU (Occurrence)  
(polyunsatd., omega-3; phosphatidylcholine  
fatty acid composition changes association with altered skeletal muscle  
insulin  
responsiveness in normal man)

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 7 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:521761 HCAPLUS

DOCUMENT NUMBER: 131:308697

TITLE: Salicylhydroxamic acid inhibits  $\Delta 6$  desaturation  
in the microalga *Porphyridium cruentum*

AUTHOR(S): Khozin-Goldberg, I.; Bigogno, C.; Cohen, Z.

CORPORATE SOURCE: The Laboratory for Microalgal Biotechnology, Jacob  
Blaustein Institute for Desert Research, Sede-Boker  
Campus, Israel

SOURCE: Biochimica et Biophysica Acta (1999), 1439(3), 384-394  
CODEN: BBACAQ; ISSN: 0006-3002

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Treatment of the microalga *Porphyridium cruentum* with salicylhydroxamic

acid (SHAM) inhibited growth and affected fatty acid composition. At a relatively low concentration (40  $\mu$ M) SHAM predominantly inhibits  $\Delta 6$  desatn. The effect of the inhibitor was most intense in phosphatidylcholine (PC) and phosphatidylethanolamine, in which the proportions of the downstream products of the  $\Delta 6$  desaturase were reduced, whereas that of the substrate, 18:2, increased. As a result of the availability of 18:2, 18:3 $\omega$ 3, which under normal conditions is not observed, appeared predominantly in chloroplastic lipids. Pulse labeling with linoleic acid has shown that SHAM inhibits  $\Delta 6$  desatn. almost immediately, suggesting an apparent inhibition of the activity of the desaturase, rather than its synthesis or that of its cofactors. Furthermore, the addition of  $\gamma$ -linolenic acid to SHAM-inhibited cultures relieved the inhibition. Following exposure to the inhibitor, 18:3 $\omega$ 3 appeared first in chloroplastic glycolipids and only later in PC, indicating that the former are the substrates for the first dedicated step of the proposed  $\omega$ 3 pathway in this alga.

CC 10-2 (Microbial, Algal, and Fungal Biochemistry)

IT **Fatty acids, biological studies**

RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)

(polyunsatd., omega-3;  $\omega$ 3 fatty acid pathway in Porphyridium cruentum)

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines, biological studies

Phospholipids, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)

( $\omega$  3 fatty acid pathway in Porphyridium cruentum)

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 8 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1998:126918 HCAPLUS

DOCUMENT NUMBER: 128:229722

TITLE: Effects of omega-3 fatty acids on renal function and electrolyte excretion in aged persons

AUTHOR(S): Adam, Olaf; Schubert, A.; Adam, A.; Antretter, N.; Forth, W.

CORPORATE SOURCE: Walther-Straub-Institute Pharmacology Toxicology, Munich, D-80336, Germany

SOURCE: European Journal of Medical Research (1998), 3(1/2), 111-118

CODEN: EJMRFL; ISSN: 0949-2321

PUBLISHER: I. Holzapfel Publishers

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In view of  $\omega$ -3 fatty acids inhibiting prostaglandin (PG) E2 in renal vascular disease and being under evaluation in chronic inflammatory kidney diseases 1.7 g/day eicosapentaenoic acid (EPA) was given to elderly atherosclerosis patients for 4 wk. EPA enrichment coincided with an inhibition of PG biosynthesis, a transient plasma creatinine increase, and a decrease of creatinine clearance and electrolyte excretion with the urine. After 4 wk no effect of  $\omega$ -3-fatty acids on renal function was found.

CC 18-5 (Animal Nutrition)

IT **Fatty acids, biological studies**

RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(polyunsatd., n-3;  $\omega$ -3 fatty acids

effects on kidney and electrolyte excretion in elderly)

IT Fatty acids, biological studies

Hemoglobins

**Phosphatidylcholines, biological studies**

Phospholipids, biological studies

Proteins, general, biological studies

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

( $\omega$ -3 fatty acids effects on blood, kidney and electrolyte excretion in elderly)

L33 ANSWER 9 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1997:717821 HCAPLUS

DOCUMENT NUMBER: 128:7311

TITLE: Fatty acids and phosphatidylcholines in the treatment of bipolar disorder

INVENTOR(S): Stoll, Andrew L.; Severus, Wolfram E.

PATENT ASSIGNEE(S): Brigham and Women's Hospital, USA; Stoll, Andrew L.; Severus, Wolfram E.

SOURCE: PCT Int. Appl., 13 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9739759	A2	19971030	WO 1997-US6712	19970423
WO 9739759	A3	19980115		
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9727384	A1	19971112	AU 1997-27384	19970423
US 6344482	B1	20020205	US 1999-269361	19990322
US 2002091103	A1	20020711	US 2002-68035	20020205
US 2003012827	A1	20030116	US 2002-83913	20020227
PRIORITY APPLN. INFO.:			US 1996-16140P	P 19960424
			WO 1997-US6712	W 19970423
			US 1999-269361	A1 19990322

AB The present invention is directed to a method of treating patients with bipolar disorder by administering  $\omega$ -3 fatty acids. These may be administered in a substantially purified form, as part of a pharmaceutical composition, or as part of a larger mol., e.g. a triacylglycerol, which releases free fatty acid after ingestion by a patient. The present invention is also directed to triacylglycerols which are esterified at the  $\gamma$ -carbon of glycerol with phosphocholine and at either the  $\alpha$ - or  $\beta$ -carbon of glycerol with an  $\omega$ -3 fatty acid. These . **omega.-3 phosphatidylcholines** are also used in the treatment of patients with bipolar disorder.

ICM A61K033-14

ICS A61K031-66; A61K031-20

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 1

IT Fatty acids, biological studies

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(polyunsatd., n-3;  $\omega$ -3 fatty acids  
and phosphatidylcholines for treatment of bipolar disorder)

**IT Phosphatidylcholines, biological studies**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

( $\omega$ -3 fatty acids and phosphatidylcholines for  
treatment of bipolar disorder)

L33 ANSWER 10 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1997:683699 HCAPLUS

DOCUMENT NUMBER: 128:3265

TITLE: The influence of different ratios and dosages of an  
 $\omega$ 6: $\omega$ 3 fatty acid supplement on the  
lipoprotein cholesterol and fatty acid profile in  
nonhuman primates on a western atherogenic diet

AUTHOR(S): van Jaarsveld, P. J.; Smuts, C. M.; Tichelaar, H. Y.;  
Kruger, M.; Lombard, C. J.; Benade, A. J. S.

CORPORATE SOURCE: National Research Programme for Nutritional  
Intervention, Medical Research Council, Tygerberg,  
7505, S. Afr.

SOURCE: Nutrition Research (New York) (1997), 17(11/12),  
1733-1747

CODEN: NTRSDC; ISSN: 0271-5317

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vervet monkeys consuming an atherogenic diet were supplemented with either  
200 or 800 mg/d of each of four different  $\gamma$ -linolenic  
acid:eicosapentaenoic acid ratios (9:1, 4:1, 2:1, and 1:1) for 12 wk.  
Plasma and lipoprotein lipids, and low d. lipoprotein (LDL) fatty acid  
analyses were done before and after supplementation. The 4:1  
 $\omega$ 6: $\omega$ 3 fatty acid supplement at 200 mg/d elicited the most  
favorable plasma lipoprotein cholesterol response. The cholesterol  
concentration  
in LDL and high d. lipoprotein was resp. decreased and increased.  
Linoleic and arachidonic acid content in the LDL cholesteryl esters  
remained constant Dihomo- $\gamma$ -linolenic and eicosapentaenoic acid were  
increased in LDL phosphatidylcholine, while the arachidonic acid content  
remained relatively constant It is concluded that the 4:1 ratio at 200 mg/d  
was the optimum supplement in our model. The long-term effect of this  
supplement on lipoprotein metabolism and atherosclerosis needs to be  
investigated.

CC 18-5 (Animal Nutrition)

IT Fatty acids, biological studies

**Phosphatidylcholines, biological studies**

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(influence of different ratios and dosages of an  $\omega$ 6:  
 $\omega$ 3 fatty acid supplement on the lipoprotein  
cholesterol and fatty acid profile in nonhuman primates on a western  
atherogenic diet)

**IT Fatty acids, biological studies**

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)



(polyunsatd., n-3; influence of different ratios and dosages of an  $\omega$ 6: $\omega$ 3 fatty acid supplement on the lipoprotein cholesterol and fatty acid profile in nonhuman primates on a western atherogenic diet)

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 11 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1995:736247 HCAPLUS

DOCUMENT NUMBER: 123:226860

TITLE: Influence of hypothyroid state on cardiac sarcolemmal incorporation of dietary  $\omega$ -6 and  $\omega$ -3 fatty acids

AUTHOR(S): Pehowich, D. J.; Awumey, E. M. K.

CORPORATE SOURCE: Departments of Oral Biology and Medicine, University of Alberta, Edmonton, AB, T6G 2N8, Can.

SOURCE: Nutrition Research (New York) (1995), 15(8), 1211-22  
CODEN: NTRSDC; ISSN: 0271-5317

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Diets enriched in either  $\omega$ -6 or long-chain  $\omega$ -3 fatty acids were fed to euthyroid and hypothyroid weanling rats. Feeding euthyroid animals the  $\omega$ -3 diet significantly reduced the  $\omega$ -6/ $\omega$ -3 ratio in all phospholipids from cardiac sarcolemma, except sphingomyelin, relative to  $\omega$ -6-fed animals. The content of 20:4 $\omega$ 6 was reduced in all phospholipid classes in  $\omega$ -3-fed animals indicating a suppression of  $\Delta$ 6- and  $\Delta$ 5-desaturase activity. Membrane levels of 20:5 $\omega$ 3, 22:5 $\omega$ 3 and 22:6 $\omega$ 3 were higher in  $\omega$ -3-fed animals with the result that the total  $\omega$ -3 content of all phospholipid classes was increased several-fold, again except in sphingomyelin. Four weeks after the induction of hypothyroidism the  $\omega$ -6/ $\omega$ -3 ratio was not different from euthyroid animals, regardless of diet. However, the 20:4/18:2 ratio was lowered even more indicating a further decrement in  $\Delta$ 6- and  $\Delta$ 5-desaturase activity. The incorporation of dietary long-chain  $\omega$ -3 fatty acids into sarcolemmal phospholipids was not affected by the hypothyroid state. The data suggests that in growing rats thyroid status affects the influence dietary fatty acids have on cardiac sarcolemmal phospholipid fatty acid compns., primarily through effects on incorporation and metabolism of  $\omega$ -6 and  $\omega$ -3 fatty acids. These changes have the potential to modulate the activities of membrane functions associated with myocardial performance.

CC 18-5 (Animal Nutrition)

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Phospholipids, biological studies

Sphingomyelins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(hypothyroid state effect on cardiac sarcolemmal incorporation of dietary  $\omega$ -6 and  $\omega$ -3 fatty acids)

IT **Fatty acids, biological studies**

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(polyunsatd., n-3, hypothyroid state

effect on cardiac sarcolemmal incorporation of dietary  $\omega$ -6 and

ω-3 fatty acids)

L33 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1995:278786 HCAPLUS

DOCUMENT NUMBER: 122:55011

TITLE: Dietary ω3 fatty acids and cholesterol modify enterocyte microsomal membrane phospholipids, cholesterol content and phospholipid enzyme activities in diabetic rats

AUTHOR(S): Keelan, M.; Doring, K.; Tavernini, M.; Wierzbicki, E.; Clandinin, M. T.; Thomson, A. B. R.

CORPORATE SOURCE: Dep. Med., Univ. Alberta, Edmonton, AB, T6G 2C2, Can.

SOURCE: Lipids (1994), 29(12), 851-8  
CODEN: LPDSAP; ISSN: 0024-4201

PUBLISHER: AOCS Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Diabetes-associated changes in the intestinal uptake of nutrients are modified by isocaloric variations in the type of dietary lipids and are associated with alterations in the phospholipid and fatty acyl content of the intestinal brush border membrane. The present study was designed to test the hypothesis that diet- and diabetes-associated changes in enterocyte microsomal membrane phospholipids are due to variations in the activity of 2 phospholipid-metabolizing enzymes, 1,2-diacylglycerol:CDPcholine cholinephosphotransferase (CPT) and phosphatidylethanolamine methyltransferase (PEMT). Adult female Wistar rats were fed 1 of 4 semisynthetic diets - beef tallow low in cholesterol (BT), beef tallow high in cholesterol (BTC), fish oil low in cholesterol (FO), or fish oil high in cholesterol. In half of the animals, diabetes mellitus was produced by injection of streptozotocin. Jejunal and ileal enterocyte microsomes (EMM) were isolated and analyzed for cholesterol and phospholipids, as well as for CPT and PEMT activities. In control animals, feeding FO reduced EMM total phospholipids, including phosphatidylcholine (PC), phosphatidylethanolamine (PE), and phosphatidylinositol. Feeding FO resulted in a >95% reduction in the activity of CPT. Diabetes was associated with increased jejunal EMM total phospholipids, including sphingomyelin (SM) and PE, without associated changes in CPT or PEMT. Dietary cholesterol supplementation did not affect EMM total cholesterol or phospholipid composition in control rats fed BT or FO, but was associated with an increase in EMM cholesterol in diabetic rats fed BT or FO. A decrease in total phospholipids due to a decline in SM, PC, and PE in diabetic rats fed FO was not associated with changes in the activities of CPT or PEMT in EMM. Thus, enterocyte microsomal membrane cholesterol and phospholipid contents are influenced by diabetes, dietary cholesterol, and the type of fatty acid in the diet, and changes in phospholipid composition are not fully explained by alterations in the activities of CPT and PEMT.

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 14

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phospholipids, biological studies

Sphingomyelins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(dietary ω 3 fatty acids and cholesterol  
modify enterocyte microsomal membrane phospholipids, cholesterol  
content and phospholipid enzyme activities in diabetic rats)

**IT Fatty acids, biological studies**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)  
 (polyunsatd., n-3, dietary  $\omega$ 3 fatty acids and cholesterol modify enterocyte microsomal membrane phospholipids, cholesterol content and phospholipid enzyme activities in diabetic rats)

L33 ANSWER 13 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1995:109112 HCAPLUS

DOCUMENT NUMBER: 122:30465

TITLE: Development of visual acuity in relation to plasma and erythrocyte  $\omega$ -6 and  $\omega$ -3 fatty acids in healthy term gestation infants

AUTHOR(S): Innis, Sheila M.; Nelson, Carolanne M.; Rioux, M France; King, D Janette

CORPORATE SOURCE: Department of Paediatrics, University of British Columbia, Vancouver, BC, V5Z 4H4, Can.

SOURCE: American Journal of Clinical Nutrition (1994), 60(3), 347-52

CODEN: AJCNAC; ISSN: 0002-9165

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The development of preferential looking acuity was studied prospectively to 3 mo of age in exclusively breast-fed and formula-fed term gestation infants. The formula contained (% of total fatty acids) 17.9% linoleic acid (18:2 $\omega$ -6) and 2.1%  $\alpha$ -linolenic acid (18:3 $\omega$ -3) but no docosahexaenoic acid (22:6 $\omega$ -3) or arachidonic acid (20:4 $\omega$ -6). The breast milk contained ( $\chi \pm$  SEM) 13.4  $\pm$  0.8% 18:2 $\omega$ -6, 1.5  $\pm$  0.1% 18:3 $\omega$ -3, 0.51  $\pm$  0.03% 20:4 $\omega$ -6, and 0.22  $\pm$  0.02% 22:6 $\omega$ -3. There were no significant differences in acuity at 14 d or 3 mo, despite substantial differences in erythrocyte and plasma lipid 22:6 $\omega$ -3. Visual acuity was [ $\chi$  (cycles/degree)  $\pm$  SD (octaves)] 3.93  $\pm$  0.54 and 4.77  $\pm$  0.48 and erythrocyte phosphatidylethanolamine %22:6 $\omega$ -3 was ( $\chi \pm$  SE) 7.6  $\pm$  0.5 and 4.0  $\pm$  0.2 in the 3-mo-old breast-fed and formula-fed infants, resp. These studies show that feeding formula containing 2.1% 18:3 $\omega$ -3 ( $\approx$  1.0% energy) results in development of visual acuity similar to breast-feeding in term infants to  $\geq$  3 mo of age.

CC 18-5 (Animal Nutrition)

**IT Phosphatidylcholines, biological studies**

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(visual acuity in relation to  $\omega$ -6 and  $\omega$ -3 fatty acid status in phosphatidylcholines of healthy infants)

**IT Fatty acids, biological studies**

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(polyunsatd., n-3, development of visual acuity in relation to  $\omega$ -6 and  $\omega$ -3 fatty acid status in healthy term gestation infants)

L33 ANSWER 14 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1994:215870 HCAPLUS

DOCUMENT NUMBER: 120:215870

TITLE: Effects of two Artemia diets with different contents

- of polyunsaturated fatty acids on the lipid composition of larvae of Atlantic herring (*Clupea harengus*)
- AUTHOR(S):** Navarro, J. C.; Batty, R. S.; Bell, M. V.; Sargent, J. R.
- CORPORATE SOURCE:** Sch. Nat. Sci., Stirling Univ., Stirling, FK9 4LA, UK
- SOURCE:** Journal of Fish Biology (1993), 43(4), 503-15  
CODEN: JFIBA9; ISSN: 0022-1112
- DOCUMENT TYPE:** Journal
- LANGUAGE:** English
- AB** Atlantic herring larvae were fed 2 enriched *Artemia* diets with different contents of (n-3) highly unsatd. fatty acids (HUFA), 1 containing low levels of 20:5(n-3) and no 22:6(n-3), the other containing substantial levels of both 20:5(n-3) and 22:6(n-3). After 30 days of culture, fatty acid compns. of lipid classes in the heads, bodies, and eyes of the larvae were analyzed. Fish fed *Artemia* with the low (n-3)HUFA diet lacking 22:6(n-3) had less total (n-3)HUFA and, in particular, of 22:6(n-3) in individual phospholipids and total neutral lipids of heads, bodies, and eyes than fish fed *Artemia* with high levels of (n-3)HUFA. The amount of 22:6(n-3) in the fatty acids of phosphatidylethanolamine of eyes was particularly susceptible to dietary depletion. The implications of these findings are discussed, particularly in relation to dietary requirements for 22:6(n-3) during development of neural tissue in predatory fish larvae.
- CC** 18-5 (Animal Nutrition)
- IT** Lipids, biological studies  
**Phosphatidylcholines, biological studies**  
Phosphatidylethanolamines  
**RL:** BIOL (Biological study)  
(omega-3 fatty acids of brine shrimp in feed effect on, of herring)
- IT** Fatty acids, biological studies  
**RL:** BIOL (Biological study)  
(polyunsatd., n-3, of herring lipids, n-3 fats of brine shrimp in feed effect on)
- L33** ANSWER 15 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN
- ACCESSION NUMBER:** 1993:516225 HCAPLUS
- DOCUMENT NUMBER:** 119:116225
- TITLE:** Comparative effects of eicosapentaenoic acid and docosahexaenoic acid on n-6 and n-3 fatty acid profiles of phospholipid classes in several tissues of rats fed a hypertriglyceridemic diet
- AUTHOR(S):** Taniguchi, Hironobu; Suzuki, Kaoru; Takita, Tosichika; Chung, Seung Yong; Hayakawa, Takashi; Nakamura, Kahoru; Innami, Satoshi
- CORPORATE SOURCE:** Dep. Nutr., Tokyo Univ. Agric., Tokyo, 156, Japan
- SOURCE:** Journal of Clinical Biochemistry and Nutrition (1993), 14(3), 151-62  
CODEN: JCBNER; ISSN: 0912-0009
- DOCUMENT TYPE:** Journal
- LANGUAGE:** English
- AB** Dietary hypertriglyceridemic rats were treated with refined eicosapentaenoic acid (EPA) and decosahexaenoic acid (DHA), and changes in the n-6 and n-3 polyunsatd. fatty acid (PUFA) profiles in phospholipid (PL) classes in their tissues were analyzed in various aspects. The effects of EPA on the PUFA profiles in tissue PL classes were different from those of DHA. The effects were manifested differently depending on tissues and on the PL classes even in the same tissue. A decrease in the proportion of n-6 PUFA and an increase in the proportion of n-3 PUFA were

both marked, particularly in the liver and heart, due to treatment with EPA and DHA, whereas these changes were somewhat slighter in the tests and were hardly observed in the brain. The variation pattern of tissue difference in the individual PUFA of each PL class differed, depending on the kind of PUFA, among the control, EPA, and DHA groups. The variation pattern of PL class difference in the individual PUFA of each tissue showed a similar tendency. Suppression of metabolic conversion from linoleic acid to arachidonic acid was not uniform, depending on the tissue and also on the PL class, but the effect of DHA was more intense compared with that of EPA. On the other hand, the ratio of n-3 PUFA/total PUFA was largest in phosphatidylethanolamine, followed by phosphatidylcholine and cardiolipin, in all the tissues, and was larger in the DHA group than in the EPA group. These findings suggest that a large uptake of n-3 PUFA by the liver PC and PE classes may decrease the secretion of VLDL-TG from the liver and may be related to the decrease of serum triglyceride.

CC 18-5 (Animal Nutrition)

IT Cardiolipins

**Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phospholipids, biological studies

RL: BIOL (Biological study)

( $\omega$ -3 and  $\omega$ -6-acids of, of tissues in hypertriglyceridemia, dietary eicosapentaenoic and docosahexaenoic acids effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, of phospholipid

classes of tissues in hypertriglyceridemia, dietary eicosapentaenoic and docosahexaenoic acids effect on)

L33 ANSWER 16 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1993:516220 HCAPLUS

DOCUMENT NUMBER: 119:116220

TITLE: The effect of dietary n-3 fatty acids on osmotic fragility and membrane fluidity of human erythrocytes

AUTHOR(S): Hagve, Tor Arne; Lie, O.; Groenn, M.

CORPORATE SOURCE: Natl. Hosp., Univ. Oslo, Oslo, 0027, Norway

SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation, Supplement (1993), 53(215), 75-84  
CODEN: SCLSAH; ISSN: 0085-591X

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Sixteen healthy females were randomly assigned to receive fish oil or corn oil double blind in a 28-day intervention period. Osmotic fragility of erythrocytes decreased in the fish oil-supplemented group and was not affected in the corn oil group. The decrease in osmotic fragility was maximal after 14 days and approached the pre-intervention level after 24 and 28 days of n-3 fatty acid supplementation. No change was observed in erythrocyte membrane fluidity in either of the groups. The level of n-3 fatty acids increased significantly in erythrocytes from the fish oil supplemented subjects, mainly at the expense of linoleic acid (18:2, n-6) and oleic acid (18:1). No significant changes were seen in the relative amount of arachidonic acid (20:4, n-6) or in the phospholipid/cholesterol ratio in erythrocytes, while the ratio between the sum of phosphatidylcholine and sphingomyelin/phosphatidylethanolamine (PC+SM/PE) increased during the intervention period. This study does not verify the hypothesis of a relationship between osmotic fragility and membrane fluidity. It is concluded that an increased level of n-3 fatty acids in erythrocyte membranes decreases osmotic fragility, and that this effect is

counteracted by increased PC+SM/PE ratio.

CC 18-5 (Animal Nutrition)

IT Fatty acids, biological studies

**Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phospholipids, biological studies

Sphingomyelins

RL: BIOL (Biological study)

(of human erythrocytes, dietary  $\omega$ -3 fatty acids effect on, osmotic fragility and membrane fluidity in relation to)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, osmotic fragility and membrane fluidity of human erythrocytes response to dietary)

L33 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1993:94074 HCAPLUS

DOCUMENT NUMBER: 118:94074

TITLE: Influence of  $\omega$ -3 fatty acid treatment on cardiac phospholipid composition and coronary flow of streptozocin-diabetic rats

AUTHOR(S): Black, S. C.; Katz, S.; McNeill, J. H.

CORPORATE SOURCE: Fac. Pharm. Sci., Univ. British Columbia, Vancouver, BC, V6M 1W5, Can.

SOURCE: Metabolism, Clinical and Experimental (1993), 42(3), 320-6

CODEN: METAAJ; ISSN: 0026-0495

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Cardiac effects of  $\omega$ -3 fatty acid treatment were studied in streptozotocin (STZ)-induced (55 mg/kg i.v.) diabetic male Wistar rats. Nondiabetic control and STZ-diabetic animals were treated with Promega (0.5 mL/kg/d; Warner-Lambert, Morris Plains, NJ) for a period of 4 wk beginning 2 wk after either vehicle or STZ injection. Plasma glucose, triglyceride, and cholesterol concns. were significantly elevated in diabetic animals;  $\omega$ -3 fatty acid treatment did not significantly affect these parameters. An isolated working heart preparation was used to determine aortic and coronary flow rates in control, diabetic, treated control, and treated diabetic animals. Aortic and coronary flow rates of untreated STZ-diabetic rats were significantly lower than those of controls over a range of left atrial filling pressures (7.5 to 20 cm water). Both aortic and coronary flow rates of  $\omega$ -3 fatty acid-treated diabetic animals were significantly increased above those of untreated diabetic rats. Aortic and coronary flow rates of treated diabetic rats paralleled those of control animals;  $\omega$ -3 fatty acid treatment did not affect aortic or coronary flow rates of control animals. Cardiac phosphatidylcholine (PC) and phosphatidylethanolamine (PE) and sarcoplasmic reticulum (SR) total phospholipid were isolated and the acyl composition was determined

Stearic

acid and C22:4, n-6 were significantly reduced in cardiac PE of diabetic animals. Relative to PE acyl species of untreated nondiabetic controls, treated diabetic PE had increased eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and reduced C22:4, n-6 levels. Compared with resp. untreated groups, there was a significant increase in the amount of PE EPA in  $\omega$ -3 fatty acid-treated control and diabetic animals. Alterations in acyl content of PC were restricted to  $\omega$ -3 fatty acid-treated controls; compared with untreated controls, redns. in C18:2, n-6 and C22:4, n-6 and an increase in EPA levels were found. EPA, which

is not detectable in cardiac SR of untreated animals, accounted for 1.6% and 0.7% of the total acyl species of treated control and treated diabetic animals, resp. DHA levels increased by 22% and 16% to account for 14.3% and 16.6% of  $\omega$ -3 fatty acid-treated control and diabetic SR acyl species, resp. The changes in SR DHA content were not significant. These data suggest that mechanisms in addition to membrane phospholipid alterations may be responsible for the  $\omega$ -3 fatty acid-mediated effect on aortic and coronary flow in STZ-diabetic rats.

CC 1-8 (Pharmacology)

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

RL: BIOL (Biological study)

( $\omega$  -3 fatty acid effect on, in heart,  
cardiotonic activity in relation to)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, heart phospholipid  
composition response to, in diabetes, cardiotonic activity in relation to)

L33 ANSWER 18 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1992:403017 HCAPLUS

DOCUMENT NUMBER: 117:3017

TITLE:  $\omega$ -6 and  $\omega$ -3 Fatty acids: monolayer  
packing and effects on bilayer permeability and  
cholesterol exchange

AUTHOR(S): Urquhart, R.; Chan, R. Y. S.; Li, Q. T.; Tilley, L.;  
Grieser, F.; Sawyer, W. H.

CORPORATE SOURCE: Russell Grimwade Sch. Biochem., Univ. Melbourne,  
Parkville, 3052, Australia

SOURCE: Biochemistry International (1992), 26(5), 831-41  
CODEN: BIINDF; ISSN: 0158-5231

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It has been suggested that the polyunsatd.  $\omega$ -3 fatty acid, docosahexaenoic acid (DHA), can adopt unique closely packed arrays in lipid bilayers. These conformations are predicted on the basis of mol. dynamics calcns. and are in contrast to the expanded conformations characteristic of  $\omega$ -6 unsatd. fatty acids. It has also been suggested that close packing of  $\omega$ -3 acyl chains could have a substantial affect on the phys. properties of lipid bilayers (e.g. permeability). Some exptl. tests of these predictions are reported. Surface pressure-area expts. have been carried out on DHA and its mixts. with stearic and oleic acids. At low surface pressures DHA is more expanded than oleic acid. Extrapolation to the high surface pressures characteristic of lipid bilayers indicates that the area per mol. of DHA is only marginally less than that for oleic acid. Thus there is no compelling evidence to suggest that the average area per mol. of the  $\omega$ -3 fatty acid is substantially different from the  $\omega$ -6 fatty acid at high surface pressures. Expts. also show that the permeability of bilayers to glucose and the rates of dissociation of pyrenyl cholesterol from bilayers were similar for bilayers containing DHA compared to bilayers containing

oleic acid or linoleic acid.

CC 6-6 (General Biochemistry)

IT **Phosphatidylcholines, biological studies**

RL: BIOL (Biological study)

(membrane, glucose permeability and cholesterol exchange in,  
 $\omega$  -3 and  $\omega$ -6 fatty acids effect on)

IT **Fatty acids, properties**

RL: PRP (Properties)  
 (polyunsatd., n-3, monolayer properties  
 and conformation of, bilayer permeability and cholesterol exchange in  
 relation to)

L33 ANSWER 19 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1992:150569 HCAPLUS

DOCUMENT NUMBER: 116:150569

TITLE: Does a threshold for the effect of dietary omega-3 fatty acids on the fatty acid composition of nuclear envelope phospholipids exist?

AUTHOR(S): Venkatraman, J. T.; Toohey, T.; Clandinin, M. T.

CORPORATE SOURCE: Dep. Foods Nutr., Univ. Alberta, Edmonton, AB, T6G 2C2, Can.

SOURCE: Lipids (1992), 27(2), 94-7  
 CODEN: LPDSAP; ISSN: 0024-4201

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Existence of a dietary maximum level or threshold for incorporation of  $\omega$ 3 fatty acids into membrane phospholipids is of interest as it may further define understanding of the dietary requirement for  $\omega$ 3 fatty acids. To test whether feeding increasing levels of dietary  $\omega$ 3 fatty acids continues to increase membrane  $\omega$ 3 fatty acid content, weanling rats were fed a nutritionally adequate semipurified diet which provided increasing amts. of C20 and C22 $\omega$ 3 fatty acids, such as 20:5 $\omega$ 3, 22:5 $\omega$ 3, and 22:6 $\omega$ 3. Dietary 20:5 $\omega$ 3 and 22:6 $\omega$ 3 were provided by substituting a purified shark oil concentrate of high 22:6 $\omega$ 3 content for safflower oil high in 18:2 $\omega$ 6. After 4 wk of feeding, nuclear envelopes from 4 animals in each diet group were prepared, lipid was extracted, and phospholipids separated. Arachidonic acid content

in membrane phosphatidylcholine, phosphatidylethanolamine, phosphatidylinositol, and phosphatidylserine was significantly reduced by feeding increased dietary levels of  $\omega$ 3 fatty acids. Decline of 20:4 $\omega$ 6 level in phospholipid tended to stabilize when the dietary content of total  $\omega$ 3 fatty acids reached 4-5% of total fatty acids. Above this level, dietary  $\omega$ 3 fatty acids did not result in a further decrease in membrane content of 20:4 $\omega$ 6. Increase in membrane phospholipid content of 20:5 $\omega$ 3 occurred as the dietary intake of  $\omega$ 3 fatty acids increased from 1.1% to 5% of total fatty acids. A dietary  $\omega$ 3 fatty acid level of 2.2-3% was sufficient to result in maximum incorporation of 22:6 $\omega$ 3 into membrane phosphatidylcholine and phosphatidylethanolamine, but not into phosphatidylinositol or phosphatidylserine.

CC 18-5 (Animal Nutrition)

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Phospholipids, biological studies

RL: BIOL (Biological study)

(fatty acids of, of nuclear envelope, dietary omega-3 fatty acids effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, fatty acids of nuclear envelope phospholipids response to dietary)

L33 ANSWER 20 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN



ACCESSION NUMBER: 1991:630512 HCAPLUS  
 DOCUMENT NUMBER: 115:230512  
 TITLE: Method for preparation of phospholipids with selected  
 carboxylic acid residues in the 2-position  
 INVENTOR(S): Ekstrand, Bo; Eriksson, Caj; Holmberg, Krister;  
 Oesterberg, Eva  
 PATENT ASSIGNEE(S): Berol Nobel AB, Swed.  
 SOURCE: PCT Int. Appl., 16 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9100918	A1	19910124	WO 1990-SE481	19900704

W: CA, JP, US

RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE

PRIORITY APPLN. INFO.: WO 1989-SE409 19890712

AB Phospholipids, including those containing  $\omega$ -3 unsatd. fatty acids at the 2-position, are prepared from lysophospholipids using phospholipase A2 in a water-in-oil microemulsion. Lysophosphatidylcholine 4.0 and  $\omega$ -3 unsatd. fatty acids 4.0 in isooctane 87.3 containing Na dioctyl sulfosuccinate 3.4 and an aqueous buffer pH 8.2 1.3% was incubated with phospholipase A2 (2.5 + 104 units/g lysophospholipid) at 30° for 16 h, under N. Anal. of the phospholipid indicated that >90% of the phospholipid contained the unsatd. fatty acid.

IC ICM C12P007-64

ICS C11C003-08

CC 16-2 (Fermentation and Bioindustrial Chemistry)

Section cross-reference(s): 17

IT **Lysophosphatidylcholines**

Lysophosphatidylethanolamines

Lysophosphatidylserines

RL: BIOL (Biological study)

(phospholipids containing  $\omega$  -3 unsatd. fatty acids

prepared from, with phospholipase A2 and microemulsions)

IT **Fatty acids, biological studies**

RL: PREP (Preparation)

(polyunsatd., n-3, C10-C22, phospholipids

containing, preparation of, from lysophospholipids, phospholipase A2 and microemulsions in)

L33 ANSWER 21 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1990:590234 HCAPLUS

DOCUMENT NUMBER: 113:190234

TITLE: Effect of increasing the level of  $\omega$ -3 fatty acids on rat skeletal muscle sarcoplasmic reticulum

AUTHOR(S): Stubbs, C. D.; Kisieleski, A. E.

CORPORATE SOURCE: Dep. Pathol. Cell Biol., Thomas Jefferson Univ., Philadelphia, PA, 19107, USA

SOURCE: Lipids (1990), 25(9), 553-8

CODEN: LPDSAP; ISSN: 0024-4201

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effect of dietary supplementation with fish oil as compared to corn oil on the lipid dynamics and calcium ATPase activity of rat skeletal sarcoplasmic reticulum was examined After 4 wk supplementation with fish

oil, the levels of eicosapentaenoic (20:5 $\omega$ 3), docosapentaenoic (22:5 $\omega$ 3) and docosahexaenoic (22:6 $\omega$ 3) acids in the total lipids were 5.3, 5.5, and 28.1% of the total fatty acids, resp. In contrast, with corn oil only 22:6 was found (8.9%). The level of these fatty acids in phosphatidylethanolamine from the membranes of animals fed fish oil was 4.2 (20:5), 5.4 (22:5), and 49.1% (22:6); and for phosphatidylcholine it was 5.4 (20:5), 4.6 (22:5), and 17.4% (22:6). Again, in corn oil-fed animals, only 22:6 was found in appreciable amts., namely 28.3% in phosphatidylethanolamine and 1.8% in phosphatidylcholine. The steady state fluorescence anisotropy of 1,6-diphenyl-1,3,5-hexatriene (DPH) was used to assess lipid order and was found to be only slightly less for membranes from animals supplemented with fish oil (0.120) as compared to those supplemented with corn oil (0.124). The calcium ATPase was found to be unaffected by supplementation consistent with the observed modest changes in lipid order as well as with suggestions that the enzyme is relatively insensitive to the level of unsatn. It could be argued that if large increases in fatty acyl polyunsatn. in mammalian cell membranes would lead to marked alterations in bulk membrane lipid motional properties, this may not be in the interest of preserving physiol. function. The complex mixture of phospholipid mol. species present in natural membranes may buffer against this by a type of passive adaptation, without the expenditure of metabolite energy, thus providing a homeoviscous environment able to optimally support membrane protein function.

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 13

IT Lipids, biological studies

**Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phospholipids, biological studies

RL: BIOL (Biological study)

(of muscle sarcoplasmic reticulum, dietary  $\omega$  -3 fatty acids effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, ATPase and lipids of muscle sarcoplasmic reticulum response to dietary)

L33 ANSWER 22 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1990:528359 HCAPLUS

DOCUMENT NUMBER: 113:128359

TITLE: A comparison of the effects of linolenic (18:3 $\omega$ 3) and docosahexaenoic (22:6 $\omega$ 3) acids on phospholipid bilayers

AUTHOR(S): Ehringer, William; Belcher, Daniel; Wassall, Stephen R.; Stillwell, William

CORPORATE SOURCE: Dep. Biol., Indiana Univ. Purdue Univ., Indianapolis, IN, 46205, USA

SOURCE: Chemistry and Physics of Lipids (1990), 54(2), 79-88  
CODEN: CPLIA4; ISSN: 0009-3084

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Linolenic (18:3 $\Delta$ 9,12,15) acid and docosahexaenoic (22:6 $\Delta$ 4,7,10,13,16,19) acid (DHA) may be incorporated into membranes. Effects of DHA and its metabolic precursor linolenic acid on membrane fluidity, fusion, and permeability are compared. The fatty acids were investigated as both free fatty acids and mixed-chain 18:0,18:3 and 18:0,22:6 phosphatidylcholines (PCs). Fluorescence polarization of 1,6-diphenyl-1,3,5-hexatriene and a series of anthracene stearic acid

probes indicates 20 mol incorporation of either fatty acid into dipalmitoylphosphatidylcholine bilayers broadens and depresses the temperature of the phase transition, but has almost no effect on fluidity of the liquid crystalline state. Similar fluidity was also observed in the liquid crystalline bilayers

of the mixed chain PCs using the same set of fluorescent fatty acid probes. In contrast, DHA as a free fatty acid or as part of a mixed chain PC, causes a much greater enhancement than linolenic acid of the rates of fusion and permeability as monitored by fluorescence resonance energy transfer and aqueous compartment mixing (fusion) and by lipid vesicle swelling in isotonic erythritol (permeability). These expts. establish a clear distinction between the effects of linolenic acid and DHA in membranes.

CC 6-6 (General Biochemistry)

IT **Phosphatidylcholines, biological studies**

RL: BIOL (Biological study)  
(membrane,  $\omega$ -3 fatty acids effect on properties of)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)  
(polyunsatd., n-3, phospholipid membrane properties response to)

IT **2644-64-6 4539-70-2, Distearoyl phosphatidylcholine**

17041-44-0 56421-10-4 59403-52-0

RL: BIOL (Biological study)  
(membrane,  $\omega$ -3 fatty acids effect on properties of)

L33 ANSWER 23 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1990:234283 HCAPLUS

DOCUMENT NUMBER: 112:234283

TITLE: Comparative effects of equivalent intakes of 18:3 (n-3) and of marine (n-3) fatty acids on rat cardiac phospholipid contents and fatty acid compositions

AUTHOR(S): Javouhey, Anne; Rocquelin, Gerard; Rochette, Luc; Juaneda, Pierre

CORPORATE SOURCE: Stn. Rech. Qualite Aliments Homme, INRA, Dijon, 21034, Fr.

SOURCE: Nutrition Research (New York, NY, United States) (1990), 10(3), 291-301

CODEN: NTRSDC; ISSN: 0271-5317

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Three groups of male Sprague-Dawley rats were fed for 4 wk purified diets containing 15% by weight of oil mixts. varying in the nature and content of (n-3)

polyunsatd. fatty acids (PUFA) but supplying similar levels of 18:2(n-6) (10% of the total dietary fatty acids) and of saturated fatty acids (19% of the total fatty acids). The first diet (low 18:3) contained small amts. of 18:3 (0.5% of the total fatty acids), the second (18:3) contained linolenic acid (10% of the total fatty acids) as the only source of (n-3) PUFA, and the third one (LC (n-3)) contained the same amount of long-chain (n-3) PUFA (mainly 20:5 and 22:6). Heart phospholipid classes were separated by HPLC, quantified and converted to Me esters which were analyzed by GLC using glass capillary columns. Dietary consumption of LC n-3 PUFA resulted in a much higher incorporation of 20:5n-3 and 22:6n-3 and a much lesser proportion of 20:4n-6 in heart phosphatidylcholine (PC) and phosphatidylethanolamine (PE) than an equivalent intake of 18:3n-3. Dietary LC n-3 PUFA, but not 18:3, reduced the 18:2n-6 level and augmented the (n-3) PUFA in heart diphosphatidylglycerol (DPG). Long-chain saturated and

(n-3) PUFA of sphingomyelin (SPH) are resp. decreased and increased in rats fed marine (n-3) PUFA. Finally, significant increases of the unsatn. index of PC, PE, DPG, and SPH were also seen in rats fed (n-3) PUFA enriched diets. Functional consequences of such fatty acid alterations are discussed.

CC 18-5 (Animal Nutrition)

IT Cardiopolins

Fatty acids, biological studies

Lipids, biological studies

Lysophospholipids

**Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Phospholipids, biological studies

Sphingomyelins

RL: BIOL (Biological study)

(of heart, dietary  $\omega$  -3 fatty acids and octadecatrienoic acid effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, phospholipid content

and fatty acid composition of heart response to dietary, octadecatrienoic acid in relation to)

L33 ANSWER 24 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1990:177384 HCAPLUS

DOCUMENT NUMBER: 112:177384

TITLE: Dietary supplementation with oils rich in (n-3) and (n-6) fatty acids influences in vivo levels of epidermal lipoxygenase products in guinea pigs

AUTHOR(S): Miller, Craig C.; Ziboh, Vincent A.; Wong, Teresa; Fletcher, Mark P.

CORPORATE SOURCE: Dep. Dermatol., Univ. California, Davis, CA, 95616, USA

SOURCE: Journal of Nutrition (1990), 120(1), 36-44

CODEN: JONUAI; ISSN: 0022-3166

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Certain dietary oils may have therapeutic potential in the treatment of inflammatory skin disorders. Presumably, the fatty acid constituents of these dietary oils exert their effects by altering the levels of cutaneous eicosanoids. Prompted by this possibility, it was investigated whether supplementation of guinea pig diets with fish oil [rich in 20:5(n-3)] or borage oil [rich in 18:3(n-6)] could significantly alter epidermal levels of eicosanoids compared with control animals supplemented with olive oil. After feeding periods of 4, 8, or 12 wk, the epidermis from the animals was analyzed for: (1) fatty acid composition of individual epidermal phospholipids, (2) levels of lipoxygenase products, and (3) levels of cyclooxygenase products (prostaglandins). The animals supplemented with dietary fish oil had elevated levels of 20:5(n-3) in epidermal phospholipids and elevated epidermal levels of 15-hydroxyeicosapentaenoic acid [the 15-lipoxygenase product of 20:5(n-3)] compared with guinea pigs fed olive oil or borage oil. Similarly, the animals supplemented with dietary borage oil had elevated levels of 20:3(n-6) [the epidermal elongase product of 18:3(n-6)] in epidermal phospholipids and elevated epidermal levels of 15-hydroxyeicosatrienoic acid [the epidermal 15-lipoxygenase product of 20:3(n-6)] compared with guinea pigs fed olive oil or fish body oil. There were no significant changes in epidermal

levels of prostaglandins. Both 15-hydroxyeicosapentaenoic acid and 15-hydroxyeicosatrienoic acid have been identified as possible anti-inflammatory metabolites, and their elevated presence in the epidermis of animals fed oils rich in 20:5(n-3) or 18:3(n-6) may provide a mechanism for the beneficial effects of these oils on inflammatory conditions.

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 1

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Phospholipids, biological studies

RL: BIOL (Biological study)

(fatty acids of, of skin epidermis, dietary oils rich in  
 $\omega$ -3 and  $\omega$ -6 fatty acids effect on, skin  
inflammation treatment in relation to)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, lipoxygenase products  
of epidermis response to dietary, skin inflammation therapy in relation  
to)

L33 ANSWER 25 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1990:20281 HCAPLUS

DOCUMENT NUMBER: 112:20281

TITLE: Effects of dietary n-3 fatty acids on mass changes and  
[3H]glycerol incorporation in various glycerolipid  
classes of rat kidney in vivo

AUTHOR(S): Yeo, Young K.; Philbrick, Diana J.; Holub, Bruce J.

CORPORATE SOURCE: Dep. Nutr. Sci., Univ. Guelph, Guelph, ON, N1G 2W1,  
Can.

SOURCE: Biochimica et Biophysica Acta (1989), 1006(1), 9-14  
CODEN: BBACAQ; ISSN: 0006-3002

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of dietary fish oil containing n-3 polyunsatd. fatty acids on triacylglycerol synthesis and phospholipid metabolism (including the alkylacyl subclass of choline glycerophospholipids (CGP)) were studied in rat kidney in vivo. After a 3-wk feeding period, the triacylglycerol content (in  $\mu$ mol/g kidney) was 47% lower in the fish oil group relative to animals given sunflower oil. This alteration was accompanied by a substantially lower amount of arachidonic acid (20:4(n-6)) and higher level ( $\mu$ mol/g tissue) of eicosapentaenoic acid (20:5(n-3)) plus docosahexaenoic acid (22:6(n-3)) in the triacylglycerol, CGP, and ethanolamine glycerophospholipids (EGP) of the fish oil group. The labeling of triacylglycerol relative to phospholipid from [3H]glycerol following i.p. administration was 49% lower in the fish oil as compared to the sunflower oil group, indicating a suppression of renal triacylglycerol synthesis relative to phospholipid synthesis. Modest differences in the labeling of CGP and EGP were found. A moderate and significantly lower proportional labeling (by 35%) of the alkylacyl subclass of CGP was observed in the fish oil as compared to the sunflower oil animals. These findings may have relevance to eicosanoid and platelet-activating factor (PAF) biosyntheses as well as renal function and pathophysiol.

CC 18-5 (Animal Nutrition)

IT **Glycerides, biological studies**

Phosphatidalcholines

Phosphatidaethanolamines

**Phosphatidylcholines, biological studies**  
Phosphatidylethanolamines  
Phospholipids, biological studies  
RL: FORM (Formation, nonpreparative)  
(formation of, by kidney, dietary  $\omega$ -3 fatty acids effect on)

IT **Phosphatidylcholines, biological studies**  
Phosphatidylethanolamines  
RL: FORM (Formation, nonpreparative)  
(alkyl analogs, formation of, by kidney, dietary  $\omega$ -3 fatty acids effect on)

IT **Fatty acids, biological studies**  
RL: BIOL (Biological study)  
(polyunsatd., n-3, glycerolipid formation by kidney response to dietary)

L33 ANSWER 26 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1989:211456 HCAPLUS

DOCUMENT NUMBER: 110:211456

TITLE: Menhaden fish oil, n-3 polyunsaturated fatty acids, and protection of newborn rats from oxygen toxicity

AUTHOR(S): Sosenko, Ilene R. S.; Innis, Sheila M.; Frank, Lee

CORPORATE SOURCE: Sch. Med., Univ. Miami, Miami, FL, 33101, USA

SOURCE: Pediatric Research (1989), 25(4), 399-404

CODEN: PEREBL; ISSN: 0031-3998

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Newborn rats born to mothers fed a high n-6 polyunsatd. fatty acid (PUFA) (safflower oil) diet demonstrated increased n-6 PUFA in lung lipids and superior tolerance to high O<sub>2</sub> exposure. Thus, it was explored whether high n-3 PUFA might also protect against hyperoxic damage and by what mechanism. Adult female rats were fed either regular rat chow, a high n-3 (menhaden fish oil-based) diet, or a high n-6 (safflower oil-based) diet for 6-wk before and then throughout pregnancy and lactation. Newborn offspring of the high n-3 (fish oil) dams demonstrated increased n-3 PUFA (i.e., eicosapentaenoic and docosahexaenoic acid) and decreased n-6 PUFA (i.e., linoleic and arachidonic acid) in their lung lipids compared to the other two diet groups. The high n-6 (safflower oil) offspring had the opposite PUFA lung lipid pattern (with increases in total n-6 fatty acids and decreases in total n-3 fatty acids). The high n-3 offspring demonstrated markedly decreased lung levels of PGE<sub>2</sub>, PGF<sub>2</sub> $\alpha$ , and TXB<sub>2</sub>, whereas the high n-6 offspring had higher eicosanoid levels than the regular diet offspring. Offspring of both high n-6 and high n-3 diet dams demonstrated essentially the same superior hyperoxic tolerance compared to regular diet offspring survival rates of 110/115 and 99/109, resp., vs. 70/91. These studies lend further support to the speculation that increasing lung PUFA content may provide the newborn lung with increased ability to scavenge O free radicals and thus may serve to protect against O<sub>2</sub> toxicity.

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 4

IT Eicosanoids

Glycerides, biological studies

**Phosphatidylcholines, biological studies**

Phospholipids, biological studies

RL: BIOL (Biological study)

(of lung, of newborn, maternal dietary menhaden oil and  $\omega$ -3 fatty acids effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)  
(polyunsatd., n-3, oxygen toxicity to  
newborn protection by maternal dietary)

L33 ANSWER 27 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1989:191601 HCAPLUS

DOCUMENT NUMBER: 110:191601

TITLE:  $\alpha$ -Linolenic acid and long-chain  $\omega$ -3 fatty  
acid supplementation in three patients with  $\omega$ -3  
fatty acid deficiency: effect on lymphocyte function,  
plasma and red cell lipids, and prostanoid formation  
AUTHOR(S): Bjerve, Kristian S.; Fischer, Sven; Wammer, Finn;  
Egeland, Torstein

CORPORATE SOURCE: Dep. Clin. Chem., Trondheim Reg. Hosp., Trondheim,  
N-7000, Norway

SOURCE: American Journal of Clinical Nutrition (1989), 49(2),  
290-300

CODEN: AJCNAC; ISSN: 0002-9165

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Supplementation of patients with  $\alpha$ -linolenic acid deficiency with Et  
 $\alpha$ -linolenate followed by a purified fish oil (EPA-oil) began to  
normalize symptoms within 10 days. The mitogenic response in isolated  
lymphocytes was reduced, whereas the number of T lymphocytes increased  
significantly. Serum thromboxanes, urinary excretion of  
2,3-dinor-6-oxo-PGF $_{1\alpha}$  (PGI $_2$ -M), and bleeding time were unaffected.  
 $\omega$ -3 Fatty acids were shown to be essential for normal accumulation  
of erythrocyte  $\omega$ -6 acids. The dietary intake of long-chain  
 $\omega$ -3 acids required to obtain midnormal concns. of  $\omega$ -3 acids in  
plasma and erythrocyte lipids was estimated to be 350-400 mg/day (0.4% of  
calories), whereas the corresponding mean intake of  $\alpha$ -linolenic acid  
was 990 mg/day (1.0% of calories). Essential fatty acid requirement  
should be stated as grams or milligrams per day, similarly to other  
essential nutrients.

CC 18-5 (Animal Nutrition)

IT Lipids, biological studies

**Phosphatidylcholines, biological studies**

RL: BIOL (Biological study)

(fatty acids of, of blood plasma and erythrocytes of humans in  
linolenate deficiency, linolenate and long-chain  $\omega$ -  
3 fatty acid supplementation effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, lipids of blood plasma  
and erythrocytes and lymphocyte function and prostanoid formation  
response to dietary, in linolenate deficiency in humans)

L33 ANSWER 28 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1988:527789 HCAPLUS

DOCUMENT NUMBER: 109:127789

TITLE: Importance of dietary omega-3 fatty acids in retinal  
function and brain chemistry

AUTHOR(S): Connor, William E.; Neuringer, Martha

CORPORATE SOURCE: Dep. Med., Oregon Health Sci. Univ., Portland, OR, USA

SOURCE: UCLA Forum in Medical Sciences (1988), 28(Nutr.

Modulation Neural Funct.), 191-201

CODEN: UCMSAA; ISSN: 0082-7134

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Female rhesus monkeys given a semipurified diet deficient in n-3 fatty acids for 2 mo before conception and throughout pregnancy developed severe and progressive depletion of n-3 fatty acids from the blood plasma and tissues, including erythrocytes, liver, skin, adipose tissue, cerebral cortex, and retina. In particular, docosahexaenoic acid (C22:6n-3) was depleted from neural and retinal phospholipids and was replaced by the n-3 fatty acids C22:4 and C22:5. These biochem. changes were associated with significant impairments in the development of visual acuity and in the recovery of the electroretinogram.

CC 18-5 (Animal Nutrition)

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Phospholipids, biological studies

RL: BIOL (Biological study)

(fatty acids of, of brain and retina, maternal ω -  
3 fatty acid deficiency effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, brain chemical and  
retinal function in relation to dietary)

L33 ANSWER 29 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1988:509282 HCAPLUS

DOCUMENT NUMBER: 109:109282

TITLE: Effect of polyunsaturated fatty acids of the n-3 and  
n-6 series on lipid composition and eicosanoid  
synthesis of platelets and aorta and on immunological  
induction of atherosclerosis in rabbits

AUTHOR(S): Bolton-Smith, Caroline; Gibney, M. J.; Gallagher, P.  
J.; Jewell, R.; Hillier, K.

CORPORATE SOURCE: Dep. Nutr., Univ. Southampton, Southampton, UK

SOURCE: Atherosclerosis (Shannon, Ireland) (1988), 72(1),  
29-35

CODEN: ATHSBL; ISSN: 0021-9150

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effect of dietary fish oil (rich in n-3 polyunsatd. fatty acids (PUFA)), corn oil (rich in n-6 PUFA) and coconut oil (low in n-3 and n-6 PUFA) on the induction of atherosclerosis by serum sickness in rabbits was investigated over 1 yr. Dietary fish oil led to an increase in eicosapentaenoic acid (EPA) in all platelet phospholipid fractions and to a reduction in platelet phosphatidylethanolamine arachidonic acid (AA). Rabbits given fish oil showed a reduction in AA and an increase in EPA in aortic total phospholipids. Rabbits given fish oil showed lower collagen-induced platelet TXA2 release and aortic production of 6-keto-PGF1α. Serum total immune complex levels and anti-horse serum IgG levels were not influenced by diet. There was a reduction in total aortic atherosclerosis in fish oil-fed animals compared with coconut oil fed animals.

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 2

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Sphingomyelins

RL: BIOL (Biological study)



(of blood platelets, fatty acids of, dietary omega-3 and omega-6 fatty acids effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, lipid composition and eicosanoid formation in blood platelets and aorta response to dietary, atherosclerosis in relation to)

L33 ANSWER 30 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1988:453785 HCAPLUS

DOCUMENT NUMBER: 109:53785

TITLE: Effects of various combinations of  $\omega$ 3 and  $\omega$ 6 polyunsaturated fats with saturated fat on serum lipid levels and eicosanoid production in rats

AUTHOR(S): Lee, Joon Ho; Sugano, Michihiro; Ide, Takashi

CORPORATE SOURCE: Sch. Agric., Kyushu Univ., Fukuoka, 812, Japan

SOURCE: Journal of Nutritional Science and Vitaminology (1988), 34(1), 117-29

CODEN: JNSVA5; ISSN: 0301-4800

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of varying the ratio of polyunsatd./saturated fatty acids (P/S) and  $\omega$ 3/ $\omega$ 6 polyunsatd. fatty acids (PUFA) of dietary fats on lipid metabolism were studied in rats using safflower oil (SFO), linseed oil (LSO), palm oil (PLO), and a 1:1 combination of these oils. The hypocholesterolemic and hypotriglyceridemic effects depended on the P/S ratio of dietary fats, LSO ( $\omega$ 3 PUFA) being more effective than SFO ( $\omega$ 6 PUFA). A similar pattern of the response was observed on liver cholesterol and triglyceride. The liver cholesterol-lowering effect of LSO, but not SFO, remained even when they were combined with PLO. The activity of liver  $\Delta$ 6-desaturase tended to be higher while that of liver phospholipase A2 was significantly lower in the LSO group than in the SFO or PLO groups. The aortic PGI2 production and the production by platelets

of thromboxane A2 were significantly low in rats fed LSO, accompanying a distinct reduction of arachidonate in tissue phospholipids. The depressing effect of LSO disappeared when it was combined with SFO but not with PLO. There were no significant differences in enzyme activities and eicosanoid production between SFO and PLO in spite of a large difference in their P/S ratio. Thus, lipid parameters examined were complicatedly regulated by the ratios of  $\omega$ 3/ $\omega$ 6 as well as P/S, suggesting an existence of an appropriate ratio of these variables.

CC 18-5 (Animal Nutrition)

IT **Phosphatidylcholines, biological studies**

RL: BIOL (Biological study)

(of blood serum and tissues, fatty acids of, dietary  $\omega$  - 3 and  $\omega$ -6 polyunsatd. and saturated fats effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, lipids of blood and serum and tissues response to dietary)

L33 ANSWER 31 OF 31 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1988:437124 HCAPLUS

DOCUMENT NUMBER: 109:37124

TITLE: In vivo incorporation of  $\omega$ 3 fatty acids into membrane lipids of rat salivary glands and changes in adenylate-cyclase activity

AUTHOR(S): Alam, S. Q.; Alam, B. S.

CORPORATE SOURCE: Med. Cent., Louisiana State Univ., Orleans, LA, 70119,  
USA  
SOURCE: Archives of Oral Biology (1988), 33(5), 295-9  
CODEN: AOBIAR; ISSN: 0003-9969  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Dietary  $\omega$ 3 fatty acids from menhaden oil were incorporated into membrane phospholipids of submandibular salivary glands (SMSG). Eicosapentaenoic (20:5), docosapentaenoic (22:5), and docosahexaenoic (22:6) acids constituted .apprx.20% of the total fatty acids in phospholipids, phosphatidylcholine, and phosphatidylethanolamine fractions of the SMSG plasma membranes of rats fed a diet containing 10% menhaden oil for 6 wk. The changes in fatty acid composition of the membrane phospholipids were accompanied by higher adenylate cyclase activity in the SMSG membranes of rats fed 10% menhaden oil than in rats fed 10% corn oil or 8% coconut oil +2% corn oil. However, there were no diet-related differences in the fold-stimulation of adenylate cyclase activity.

CC 18-5 (Animal Nutrition)

Section cross-reference(s): 13

IT **Phosphatidylcholines, biological studies**

Phosphatidylethanolamines

Phospholipids, biological studies

RL: BIOL (Biological study)

(of salivary gland plasma membrane, fatty acids of, dietary

$\omega$  -3 fatty acids of menhaden oil effect on)

IT **Fatty acids, biological studies**

RL: BIOL (Biological study)

(polyunsatd., n-3, salivary gland

membrane lipids uptake of dietary, from menhaden oil, adenylate cyclase

of salivary gland in relation to)

↙

E14	21868	-->	Phosphatidylcholines/CT
E15	24438	MN	D10.570.755.375.760.400.800./CT
		DC	an INDEX MEDICUS major descriptor
		NOTE	Derivatives of phosphatidic acids in which the phosphoric acid is bound in ester linkage to a choline moiety. Complete hydrolysis yields 1 mole of glycerol, phosphoric acid and choline and 2 moles of fatty acids.
		INDX	/biosyn /physiol permitted
		AQ	AD AE AG AI AN BI BL CF CH CL CS CT DF DU EC GE
			HI IM IP ME PD PH PK PO RESD SE ST TO TU UR
		HNTE	77; was PHOSPHATIDYL CHOLINE see under LECITHINS 1963-76; LECITHINS was heading 1963-76
		ONTE	use PHOSPHATIDYLCHOLINES to search PHOSPHATIDYL CHOLINE & LECITHINS 1966-76
		MHTH	NLM (1975)
E16	0	UF	Choline Glycerophospholipids/CT
E17	0	UF	Choline Phosphoglycerides/CT
E18	0	UF	Glycerophospholipids, Choline/CT
E19	0	UF	<del>Lecithin</del> /CT
E20	0	UF	<del>Lecithins</del> /CT
E21	0	UF	Phosphatidylcholine/CT
E22	0	UF	Phosphoglycerides, Choline/CT
E23	1858	NT1	1,2-Dipalmitoylphosphatidylcholine/CT
E24	1825	NT1	Dimyristoylphosphatidylcholine/CT
***** END *****			

UF = use for

"Phosphatidylcholines" is the preferred term for lecithin in the MESH.

=> d que

L10 7 SEA FILE=HCAPLUS ABB=ON PLU=ON LECITHIN?(5A)METAB?(S)(PHOSPHA  
TIDYLCHOLIN? OR PHOSPHATIDYL CHOL?)  
L11 6 SEA FILE=HCAPLUS ABB=ON PLU=ON LECITHIN?(S)METAB?(5A)(PHOSPHA  
TIDYLCHOLIN? OR PHOSPHATIDYL CHOL?)  
L12 8 SEA FILE=HCAPLUS ABB=ON PLU=ON L10 OR L11

=> d l12 ibib abs hitind 1-8

L12 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:392447 HCAPLUS

DOCUMENT NUMBER: 139:213427

TITLE: Prevention by dietary (n-6) polyunsaturated  
phosphatidylcholines of intrahepatic cholestasis  
induced by cyclosporine A in animals

AUTHOR(S): Chanussot, Francoise; Benkoel, Liliane

CORPORATE SOURCE: Faculte de Medecine, INSERM U. 476, Marseille, 13385,  
Fr.

SOURCE: Life Sciences (2003), 73(4), 381-392

CODEN: LIFSAK; ISSN: 0024-3205

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Dietary n-6 polyunsatd. phosphatidylcholines (vegetable lecithin) can efficiently prevent intrahepatic cholestasis induced by cyclosporine A in rats. Mechanistic studies showed that the expression of Na<sup>+</sup>,K<sup>+</sup>-ATPase, Ca<sup>2+</sup>,Mg<sup>2+</sup>-ATPase, and F-actin in the rat liver were decreased by the drug and enhanced by n-6 lecithin-enriched diet. There is a possible direct effect of **phosphatidylcholines**, vectors of polyunsatd. fatty acids, provided by the **metab.** of the dietary **lecithin**, on the hepatic parameters. The drug and diet effects may result in opposite modifications of membrane composition and fluidity. The final resp. outcomes are decreased and enhanced bile lipid secretion by cyclosporin A and vegetable lecithin enriched diet. A hypothesis of a bypass process including sep. and functional actin-independent way for the non micellar and phospholipid-dependent secretion of bile lipids is presented. The relationships between the ATPases, the microfilament components (F-actin), and different transporters still remain to be clarified. One can speculate on beneficial effects in humans of diets enriched in vegetable lecithins that might prevent cholestasis induced by cyclosporin A.

CC 18-0 (Animal Nutrition)

Section cross-reference(s): 1

REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2000:828207 HCAPLUS

DOCUMENT NUMBER: 134:70869

TITLE: Dietary soybean phosphatidylcholines lower lipidemia:  
mechanisms at the levels of intestine, endothelial  
cell, and hepato-biliary axis

AUTHOR(S): Mastellone, I.; Polichetti, E.; Gres, S.; de la

Maisonneuve, C.; Domingo, N.; Marin, V.; Lorec, A.-M.;  
Farnarier, C.; Portugal, H.; Kaplanski, G.; Chanussot,

F.  
CORPORATE SOURCE: Hopital Sainte Marguerite, INSERM U. 476, Marseille, Fr.  
SOURCE: Journal of Nutritional Biochemistry (2000), 11(9), 461-466  
CODEN: JNBIEL; ISSN: 0955-2863  
PUBLISHER: Elsevier Science Inc.  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The beneficial metabolic effects of dietary soybean lecithin on lipid metabolism are now more clearly established. The intestinal absorption of cholesterol is decreased by soybean phosphatidylcholine-enriched diet and results in a cholesterol-lowering effect. There is an enhancement of the cholesterol efflux by endothelial cells incubated with soybean phosphatidylcholines, and a stimulation of the reverse cholesterol transport by high d. lipoprotein-phosphatidylcholines. As a result of all these processes, **phosphatidylcholines** provided by the soybean **lecithin metab.** appear to be key mols. controlling the biodynamic exchanges of lipids. They regulate homeostasis of cholesterol and fatty acids by decreasing their synthesis and promoting cholesterol oxidation into bile salts. Finally, the outcome is the increase in bile secretion of these lipids and/or their metabolite forms. Such findings constitute promising goals in the field of nutritional effects of soybean lecithin in the treatment or prevention of hyperlipidemia and related atherosclerosis.  
CC 18-5 (Animal Nutrition)  
Section cross-reference(s): 13, 14  
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 1999:632366 HCAPLUS  
DOCUMENT NUMBER: 132:21450  
TITLE: **Metabolism** of oxidized **phosphatidylcholines** formed in oxidized low density lipoprotein by **lecithin**-cholesterol acyltransferase  
AUTHOR(S): Itabe, Hiroyuki; Hosoya, Ryuta; Karasawa, Ken; Jimi, Shiro; Saku, Keihiro; Takebayashi, Shigeo; Imanaka, Tsuneo; Takano, Tatsuya  
CORPORATE SOURCE: Department of Microbiology and Molecular Pathology, Faculty of Pharmaceutical Sciences, Teikyo University, Kanagawa, 199-0195, Japan  
SOURCE: Journal of Biochemistry (Tokyo) (1999), 126(1), 153-161  
CODEN: JOBIAO; ISSN: 0021-924X  
PUBLISHER: Japanese Biochemical Society  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The possible involvement of **lecithin**-cholesterol acyltransferase (LCAT) in the **metab.** of oxidized **phosphatidylcholine** (PC) in plasma was investigated. A variety of oxidized products are formed from PC following oxidation of low d. lipoproteins (LDL). A significant increase in LDL oxidation levels in patients with familial LCAT deficiency (FLD) has been previously demonstrated by a sensitive sandwich ELISA for oxidized LDL using the monoclonal antibody DLH3 which recognizes oxidized products of PC. In the present study, the authors found that

LCAT produces various metabolites from oxidized PC and that oxidized PC mols. in LDL particles serve as substrates. When the neutral lipid fraction was separated by TLC after the incubation of oxidized 1-palmitoyl-2-[1-14C]linoleoyl PC with human plasma, a number of radioactive bands were formed in addition to cholesteryl ester. These products were not formed from native 1-palmitoyl-2-[1-14C]linoleoyl PC. Plasma from FLD patients also failed to form the addnl. products from oxidized PC. The addition of dithio-bis(nitrobenzoate) (DTNB), an LCAT inhibitor, or the inactivation of LCAT activity by treating the plasma at 56° for 30 min abolished the generation of these products from oxidized PC. The activity was recovered in the high d. lipoprotein (HDL) fraction but not in the LDL fraction separated from normal plasma. When 1-palmitoyl-2-[1-14C](9-oxononanoyl) PC and 1-stearoyl-2-[1-14C](5-oxovaleroyl) PC, PC oxidation products that contain short chain aldehydes, were incubated with human plasma, radioactive products in the neutral lipid fraction were observed on TLC. LDL containing oxidized PC was measured by sandwich ELISA

using

an anti-apolipoprotein B antibody and DLH3. The reconstituted oxidized PC-LDL particles were found to have lost their ability to bind DLH3 upon incubation with HDL, while the reactivity of the reconstituted oxidized PC-LDL remained unchanged in the presence of DTNB. These results suggest that LCAT is capable of metabolizing a variety of oxidized products of PC and preventing the accumulation of oxidized PC in circulating LDL particles.

CC 13-5 (Mammalian Biochemistry)

Section cross-reference(s): 7

ST oxidized LDL **phosphatidylcholine metab**  
**lecithin** cholesterol acyltransferase

IT Lipoproteins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(low-d., oxidized; **metab.** of oxidized

**phosphatidylcholines** formed in oxidized low d. lipoprotein by **lecithin**-cholesterol acyltransferase)

IT Blood plasma

(**metab.** of oxidized **phosphatidylcholines** formed in oxidized low d. lipoprotein by **lecithin**-cholesterol acyltransferase)

IT **Phosphatidylcholines**, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(oxidized; **metab.** of oxidized **phosphatidylcholines**

formed in oxidized low d. lipoprotein by **lecithin**-cholesterol acyltransferase)

IT 57-88-5D, Cholesterol, esters with fatty acids

RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

(formation from oxidized **phosphatidylcholines**; **metab.** of oxidized **phosphatidylcholines** formed in oxidized low d. lipoprotein by **lecithin**-cholesterol acyltransferase)

IT 9031-14-5, **Lecithin**-cholesterol acyltransferase

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(**metab.** of oxidized **phosphatidylcholines** formed in oxidized low d. lipoprotein by **lecithin**-cholesterol acyltransferase)

IT 6931-84-6D, 1-Palmitoyl-2-linoleoyl **phosphatidylcholine**,  
oxidized 186638-17-5 251927-17-0  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(**metab.** of oxidized **phosphatidylcholines** formed in  
oxidized low d. lipoprotein by **lecithin**-cholesterol  
acyltransferase)  
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 1999:508838 HCAPLUS  
DOCUMENT NUMBER: 131:226676  
TITLE: Role of lecithin-cholesterol acyltransferase in the  
metabolism of oxidized phospholipids in plasma:  
studies with platelet-activating factor-acetyl  
hydrolase-deficient plasma  
AUTHOR(S): Subramanian, Veedamali S.; Goyal, Jaya; Miwa, Masao;  
Sugatami, Junko; Akiyama, Masaki; Liu, Ming; Subbaiah,  
Papasani V.  
CORPORATE SOURCE: Departments of Medicine and Biochemistry, Rush Medical  
College, Chicago, IL, 60612, USA  
SOURCE: Biochimica et Biophysica Acta (1999), 1439(1), 95-109  
CODEN: BBACAQ; ISSN: 0006-3002  
PUBLISHER: Elsevier Science B.V.  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB To determine the relative importance of platelet-activating  
factor-acetylhydrolase (PAF-AH) and lecithin-cholesterol acyltransferase  
(LCAT) in the hydrolysis of oxidized phosphatidylcholines (OXPCs) to  
lyso-phosphatidylcholine (lyso-PC), we studied the formation and metabolism of  
OXPCs in the plasma of normal and PAF-AH-deficient subjects. Whereas the  
loss of PC following oxidation was similar in the deficient and normal  
plasmas, the formation of lyso-PC was significantly lower, and the  
accumulation of OXPC was higher in the deficient plasma. Isolated LDL  
from the PAF-AH-deficient subjects was more susceptible to oxidation, and  
stimulated adhesion mol. synthesis in endothelial cells, more than the  
normal LDL. Oxidation of 16:0-[1-14C]-18:2 PC, equilibrated with plasma PC,  
resulted in the accumulation of labeled short- and long-chain OXPCs, in  
addition to the labeled aqueous products. The formation of the aqueous  
products

decreased by 80%, and the accumulation of short-chain OXPC increased by  
110% in the deficient plasma, compared to the normal plasma, showing that  
PAF-AH is predominantly involved in the hydrolysis of the truncated OXPCs.  
Labeled sn-2-acyl group from the long-chain OXPC was not only hydrolyzed  
to free fatty acid, but was preferentially transferred to diacylglycerol,  
in both the normal and deficient plasmas. In contrast, the acyl group  
from unoxidized PC was transferred only to cholesterol, showing that the  
specificity of LCAT is altered by OXPC. It is concluded that, while  
PAF-AH carries out the hydrolysis of mainly truncated OXPCs, LCAT  
hydrolyzes and transesterifies the long-chain OXPCs.

CC 13-6 (Mammalian Biochemistry)  
IT **Phosphatidylcholines**, biological studies  
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological  
study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC  
(Process)  
(oxidized; role of **lecithin**-cholesterol acyltransferase in

**metab.** of oxidized phospholipids in platelet-activating  
factor-acetyl hydrolase-deficient plasma)

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1996:255675 HCAPLUS

DOCUMENT NUMBER: 125:6295

TITLE: Reaction of phosphatidylcholine hydroperoxide in human  
plasma: the role of peroxidase and  
lecithin:cholesterol acyltransferase

AUTHOR(S): Nagata, Yuichiro; Yamamoto, Yorihiro; Niki, Etsuo  
CORPORATE SOURCE: Res. Cent. Adv. Sci. Technol., Univ. Tokyo, Tokyo,  
153, Japan

SOURCE: Archives of Biochemistry and Biophysics (1996),  
329(1), 24-30  
CODEN: ABBIA4; ISSN: 0003-9861

PUBLISHER: Academic

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In order to elucidate the reason why phosphatidylcholine hydroperoxide is  
unstable in human plasma, 1-palmitoyl-2-linoleoylphosphatidylcholine  
hydroperoxide (PLPC-OOH) was incubated aerobically in human plasma at  
37°, and its decomposition products were measured. The major product  
was the corresponding alc. (PLPC-PH) and this reduction probably occurred by  
an enzymic process since no acceleration in ascorbate depletion and no  
significant decrease in other plasma antioxidants were observed upon addition  
of

PLPC-OOH. Cholesteryl linoleate hydroperoxide and its alc. (Ch18:2-OH)  
were also detected as minor products. Similarly, 1-stearoyl-2-  
arachidonoylphosphatidylcholine hydroperoxide gave its alc. (SAPC-OH) as a  
major product and cholesteryl arachidonate hydroperoxide and its hydroxide  
(Ch20:4-OH) as minor products. These oxidized cholesteryl esters are  
likely to be produced by the action of lecithin:cholesterol  
acyltransferase (LCAT) present in high-d. lipoprotein (HDL) since (a)  
incubation of PLPC-OH and SAPC-OH in human plasma gave Ch18:2-OH and  
Ch20:4-OH, resp., (b) isolated human HDL converted PLPC-PH to Ch18:2-OH  
and SAPC-OH to Ch20:4-OH, while isolated human low-d. lipoprotein was  
inactive for this conversion, and (c) formation of oxidized cholesteryl  
esters in plasma and HDL was inhibited by the LCAT inhibitor  
5,5'-dithiobis(2-nitrobenzoic acid). A possible beneficial role of LCAT  
for converting phosphatidylcholine hydroperoxide to cholesteryl ester  
hydroperoxide is also discussed.

CC 13-2 (Mammalian Biochemistry)

ST phosphatidylcholine hydroperoxide metab blood peroxidase acyltransferase;  
**lecithin** cholesterol acyltransferase **phosphatidylcholine**  
hydroperoxide **metab**

L12 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1992:547487 HCAPLUS

DOCUMENT NUMBER: 117:147487

TITLE: Effects of cholinergic agents on the metabolism of  
choline in muscle from *Ascaris suum*

AUTHOR(S): Arevalo, Javier I.; Saz, Howard J.  
CORPORATE SOURCE: Dep. Biol. Sci., Univ. Notre Dame, Notre Dame, IN,  
46556, USA

SOURCE: Journal of Parasitology (1992), 78(3), 387-92



CODEN: JOPAA2; ISSN: 0022-3395

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The incorporation of [methyl-14C]choline into the choline-containing compds. of *Ascaris suum* muscle and the effects of acetylcholine and its agonists, carbachol and levamisole, on this incorporation were studied. Previous expts. reported a stimulation of **phosphatidylcholine** (**lecithin**) **metab.** upon the administration of acetylcholine. Acetylcholine administered in vitro to *A. suum* muscle and body wall preps. resulted in a stimulation of phospholipase C activity that, in turn, produced an increased rate of hydrolysis of phosphatidylcholine to the corresponding diacylglyceride (DAG). The DAG, in turn, may act as a second messenger as it is required for the activation of an *A. suum* protein kinase C. Evidence presented here is in accordance with this hypothesis. The administration of cholinergics resulted in a stimulation of phosphatidylcholine turnover. Acetylcholine also stimulated isotope incorporation into glycerophosphorylcholine, presumably as a consequence of enhanced phospholipid turnover. These events appear to be associated with the ligand binding to the acetylcholine receptors of the *A. suum* muscle. Choline kinase activity is suggested to maintain the observed high ratio of phosphorylcholine to choline. The findings indicate that in the parasite's muscle phosphatidylcholine metabolism may be linked to receptor-dependent responses and subsequent signal transduction.

CC 12-2 (Nonmammalian Biochemistry)

L12 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1990:233464 HCAPLUS

DOCUMENT NUMBER: 112:233464

TITLE: Molecular species of phosphatidylcholine in abetalipoproteinemia: effect of lecithin:cholesterol acyltransferase and lysolecithin acyltransferase

AUTHOR(S): Banerji, B.; Subbaiah, P. V.; Gregg, R. E.; Bagdade, John D.

CORPORATE SOURCE: Dep. Med., Rush Med. Coll., Chicago, IL, 60612, USA

SOURCE: Journal of Lipid Research (1989), 30(12), 1907-16

CODEN: JLPRAW; ISSN: 0022-2275

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To study the role of very-low-d. lipoproteins (VLDL) and low-d. lipoproteins (LDL) in determining the mol. species composition of phosphatidylcholine

(PC) and the specificity of lecithin:cholesterol acyltransferase (LCAT) in human plasma, the authors studied the PC species composition in plasma from abetalipoproteinemic (ABL) and control subjects before and after incubation at 37°. The ABL plasma contained significantly higher percentages of sn-2-18:1 species (16:0-18:1, 18:0-18:1, and 18:1-18:1) and lower percentages of sn-2-18:2 species (16:0-18:2, 18:0-18:2, and 18:1-18:2) as well as sn-2-20:4 species (16:0-20:4, 18:0-20:4, and 18:1-20:4). Similar abnormalities were found in the PC of ABL erythrocytes, whereas the phosphatidylethanolamine of the erythrocytes was less affected. The relative contribution of various PC species towards LCAT reaction in ABL plasma was significantly different from that found in normal plasma. Thus, whereas 16:0-18:2 and 16:0-18:1 contributed, resp., 43.8% and 15.9% of the total acyl groups used for cholesterol esterification in normal plasma, they contributed, resp., 21.5% and 37.9% in ABL plasma. The relative contribution of 16:0-20:4 was also

significantly lower in ABL plasma (4.7% vs. 9.0% in normal), whereas that of 16:0-16:0 was higher (6.4% vs. 0.5%). However, the selectivity factors of various species (percent contribution/percent concentration) were not significantly different between ABL and normal plasma, indicating that the substrate specificity of LCAT is not altered in the absence of VLDL and LDL. Incubation of ABL plasma in the presence of normal VLDL or LDL resulted in normalization of its mol. species composition and in the stimulation of its LCAT activity. Addition of LDL, but not VLDL, also resulted in the activation of lysolecithin acyltransferase (LAT) activity. The incorporation of [1-14C]palmitoyl lysoPC into various PC species in the presence of LDL was similar to that observed in normal plasma, with the 16:0-16:0 species having the highest specific activity. Thus, the absence of apoB-containing lipoproteins significantly affects the mol. species composition

composition

of plasma PC as well as its metabolism by LCAT and LAT reactions.

CC 14-14 (Mammalian Pathological Biochemistry)

ST lysolecithin acyltransferase phosphatidylcholine metab  
abetalipoproteinemia; **lecithin** cholesterol acyltransferase  
**phosphatidylcholine metab** abetalipoproteinemia

IT 9027-64-9, Lysolecithin acyltransferase

RL: BIOL (Biological study)

(**phosphatidylcholine metab.** response to, of humans  
with abetalipoproteinemia, **lecithin**:cholesterol

acyltransferase in low-d. and very-low-d. lipoproteins in relation to)

IT 9031-14-5, **Lecithin**:cholesterol acyltransferase

RL: BIOL (Biological study)

(**phosphatidylcholine metab.** response to, of humans  
with abetalipoproteinemia, lysolecithin acyltransferase and low-d. and  
very-low-d. lipoproteins in relation to)

L12 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1976:2673 HCAPLUS

DOCUMENT NUMBER: 84:2673

TITLE: Phospholipid metabolism of the mammalian lung

AUTHOR(S): Ohno, Kimiyoshi; Shimojo, Tadashi

CORPORATE SOURCE: Dep. Biochem., Sapporo Med. Coll., Sapporo, Japan

SOURCE: Taisha (1974), 11(10), 1568-85

CODEN: TSHAAW; ISSN: 0372-1566

DOCUMENT TYPE: Journal; General Review

LANGUAGE: Japanese

AB A review with 78 refs. on **phosphatidylcholine metab.**

in the mammalian lung, the biosynthetic systems, and the degree of  
contribution of each of these systems to the formation of dipalmitoyl  
**lecithin**, the lung surfactant.

CC 13-0 (Mammalian Biochemistry)

=&gt; d que

L8 1150 SEA FILE=HCAPLUS ABB=ON PLU=ON LECITHIN?(S) (PHOSPHATIDYLCHOLIN? OR PHOSPHATIDYL CHOL?)

L10 7 SEA FILE=HCAPLUS ABB=ON PLU=ON LECITHIN?(5A)METAB?(S) (PHOSPHATIDYLCHOLIN? OR PHOSPHATIDYL CHOL?)

L11 6 SEA FILE=HCAPLUS ABB=ON PLU=ON LECITHIN?(S)METAB?(5A) (PHOSPHATIDYLCHOLIN? OR PHOSPHATIDYL CHOL?)

L12 8 SEA FILE=HCAPLUS ABB=ON PLU=ON L10 OR L11

L13 103 SEA FILE=HCAPLUS ABB=ON PLU=ON L8 AND METAB?

L14 8 SEA FILE=HCAPLUS ABB=ON PLU=ON L13 AND REVIEW/DT

L15 6 SEA FILE=HCAPLUS ABB=ON PLU=ON L14 NOT L12

=&gt; d ibib abs hitind 1-6

L15 ANSWER 1 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:739946 HCAPLUS

DOCUMENT NUMBER: 140:338392

TITLE: Effects of phosphatidylcholine intake on liver function and liver carcinogenesis

AUTHOR(S): Canty, David J.

CORPORATE SOURCE: Department of Nutrition and Food Studies, New York University, New York, NY, USA

SOURCE: Nutrition and Biochemistry of Phospholipids, [International Congress], 8th, Vienna, Austria, Sept. 7-10, 2002 (2003), Meeting Date 2002, 142-152. Editor(s): Szuhaj, Bernard F.; Van Nieuwenhuyzen, Willem. AOCs Press: Champaign, Ill. CODEN: 69ENBT; ISBN: 1-893997-42-1

DOCUMENT TYPE: Conference; **General Review**

LANGUAGE: English

AB A review of data on the role of dietary **phosphatidylcholine** (**lecithin**) intake in liver functions and protection from alc. injury and other toxic insults. Choline is required for normal liver functions in humans and many animal species and has multiple functions, including protection against liver carcinogenesis. Dietary sources and intakes of choline, symptoms of choline deficiency in humans and animals, and roles in Me group **metab.** and signal transduction are also examined

CC 18-0 (Animal Nutrition)

Section cross-reference(s): 14

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:913036 HCAPLUS

DOCUMENT NUMBER: 138:299257

TITLE: The use of liposomes for constructing cell models

AUTHOR(S): Oberholzer, T.; Luisi, P. L.

CORPORATE SOURCE: Institut fuer Polymere, ETH-Zentrum, Zurich, CH-8092, Switz.

SOURCE: Journal of Biological Physics (2002), 28(4), 733-744

CODEN: JBPHBZ; ISSN: 0092-0606

PUBLISHER: Kluwer Academic Publishers

DOCUMENT TYPE: Journal; **General Review**

LANGUAGE: English

AB A review. The authors illustrate here in a form of a short review some of the work developed in the authors' and other groups aiming at performing inside liposomes enzymic reactions relevant for the origin of life. The work on giant vesicles will not be considered here. The long-range goal of the authors' work with SUVs or LUVs (small unilamellar vesicles or large unilamellar vesicles) is the construction of a model minimal cell. By this the authors mean a cell-like system containing the minimal and sufficient number of macromol. components for expressing some of the basic functions of a living cell- such as protein biosynthesis, growth and self-reproduction, homeostasis based on a primitive **metab.** The authors begin describing a POPC liposomal system containing some of the enzymes of the salvage cycle for the synthesis of **lecithin**; then vesicles containing the nucleotide phosphorylase enzyme for the polymerization

of ADP

into poly(A); an oleate self-reproducing vesicular system which hosts Q $\beta$  replicase for the replication of a RNA template; a POPC systems (POPC = 1-palmitoyl-2-oleoyl-sn-**phosphatidylcholine**) hosting the elements for a polymerase chain reaction; and finally the attempts to organize inside liposomes the ribosomal system capable of the synthesis of poly(phenylalanine). This anal. of published work will be followed by the description of novel work aimed at expressing a protein (green fluorescent protein) inside liposomes. The possible development of this work and its limits will be discussed.

CC 6-0 (General Biochemistry)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:881460 HCAPLUS

DOCUMENT NUMBER: 136:166568

TITLE: Lecithin and choline: New roles for old nutrients

AUTHOR(S): Canty, David J.

CORPORATE SOURCE: Department of Nutrition and Food Studies, New York University, New York, NY, USA

SOURCE: Handbook of Nutraceuticals and Functional Foods (2001) , 423-443. Editor(s): Wildman, Robert E. C. CRC Press LLC: Boca Raton, Fla. CODEN: 69CBDJ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review. The nutritional and biochem. roles of choline (essential nutrient) and **lecithin (phosphatidylcholine)** in various health and disease states; including cardiovascular disease, reproduction and development, memory, and phys. performance, are discussed. The potential benefits are based on the functions of lecithin and choline in Me group **metab.**, cholesterol transport, acetylcholine synthesis, and cell signaling.

CC 18-0 (Animal Nutrition)

REFERENCE COUNT: 136 THERE ARE 136 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1998:592436 HCAPLUS

DOCUMENT NUMBER: 129:341015

TITLE: Regulation of lecithin cholesterol acyltransferase activity

AUTHOR(S): Jonas, Ana  
CORPORATE SOURCE: Department of Biochemistry, College of Medicine at  
Urbana-Champaign, University of Illinois, Urbana, IL,  
61801, USA  
SOURCE: Progress in Lipid Research (1998), 37(4), 209-234  
CODEN: PLIRDW; ISSN: 0163-7827  
PUBLISHER: Elsevier Science Ltd.  
DOCUMENT TYPE: Journal; **General Review**  
LANGUAGE: English  
AB A review with 156 refs. The topics discussed include key properties,  
lecithin cholesterol acyltransferase (LCAT) measurements, regulation of  
the LCAT gene, LCAT mass in plasma, LCAT deficiencies, HDL deficiencies,  
transgenic and knock-out animals, role of LCAT in reverse cholesterol  
transport and in HDL remodeling, regulation of LCAT by substrates, and  
functional regions of LCAT.  
CC 7-0 (Enzymes)  
Section cross-reference(s): 3, 13, 14  
IT Lipoproteins  
(**metabolic** disorders; regulation of lecithin cholesterol  
acyltransferase activity in relation to disease states)  
IT Lipids, biological studies  
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)  
(**metabolic** disorders; regulation of lecithin cholesterol  
acyltransferase activity in relation to disease states)  
IT Gene, animal  
**Phosphatidylcholines**, biological studies  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(regulation of **lecithin** cholesterol acyltransferase activity  
in relation to disease states)  
REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 5 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 1985:611738 HCAPLUS  
DOCUMENT NUMBER: 103:211738  
TITLE: Lecithin in health and disease  
AUTHOR(S): Zeisel, Steven H.  
CORPORATE SOURCE: Dep. Pathol., Boston Univ., Boston, MA, 02118, USA  
SOURCE: AOCS Monograph (1985), 12(Lecithins), 323-45  
CODEN: AOMODZ; ISSN: 0731-4183  
DOCUMENT TYPE: Journal; **General Review**  
LANGUAGE: English

AB A review, with 189 refs., on **lecithin** (  
**phosphatidylcholine**) dietary sources, absorption from the gut,  
**metab.**, and therapeutic usages.  
CC 13-0 (Mammalian Biochemistry)  
Section cross-reference(s): 1, 14, 18

L15 ANSWER 6 OF 6 HCAPLUS . COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 1984:207915 HCAPLUS  
DOCUMENT NUMBER: 100:207915  
TITLE: Egg yolk **lecithin**. Phospholipids and  
**phosphatidylcholine**  
AUTHOR(S): Chigira, Jun  
CORPORATE SOURCE: Asahi Kasei Kogyo Co. Ltd., Japan  
SOURCE: New Food Industry (1984), 26(3), 22-6

CODEN: NYFIAM; ISSN: 0547-0277

DOCUMENT TYPE: Journal; **General Review**

LANGUAGE: Japanese

AB A review with 19 refs., discussing characteristics, composition, **metab** ., and physiol. activities of egg and soybean lecithins. A special reference is made to the nutritional value of lecithins as a fish feed additive and human food supplement.

CC 17-0 (Food and Feed Chemistry)